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ABSTRACT

This curriculum guide on substance abuse is intended for teachers of family medicine. Comments, learning objectives, teaching hints, and evaluations of knowledge are provided for each area in all chapters. Chapter 1 focuses on the pharmacology of commonly abused drugs including depressants, opioids, stimulants, hallucinogens, inhalants, and various drug interactions. For each substance, the chemistry, metabolism, psychoactive properties, body system effects, intoxication, and tolerance and dependence levels are discussed. Chapter 2 discusses the pathophysiology of ethanol abuse. Chapter 3 deals with the identification of substance abusers, while chapter 4 addresses change motivation. Chapters 5 through 7 address detoxification, rehabilitation, and prevention. Chapters 8 and 9 address chemical dependency in the family and in the family physician, taking into consideration assessment, treatment, and education and counseling needs. The last chapter presents strategies for curricular change in the university, the medical school, family medicine departments, and community training programs. References and materials citations are appended. (BL)

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FAMILY MEDICINE CURRICULUM GUIDE TO SUBSTANCE ABUSE

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DEDICATION TO THE MEMORY OF CARLIE MARIE GOODWIN

By John E. Arradondo

It is an honor to be asked to write a dedicatory word about a colleague who was a friend. On 26 September 1982, Carlisle Marie Goodwin M.Ed., M.P.H. died young and from unknown causes. From 1969, when I first observed her people-oriented professional talents, until her death, Carlisle dedicated her personal and professional life to the betterment of humanity. I am pleased that her colleagues in the field of substance abuse will honor her memory by this dedication.

In the early seventies, while pursuing graduate studies in education, health services administration, and health education in Boston, she demonstrated her commitment to family care and community service by working with a local self-help drug abuse program to produce a series of effective audiovisual materials for client, family and community education. She continued to use her professional skills to illustrate her commitment in Nashville, her last residence, where she inaugurated an adolescent care program in a local health center, a teen health education program in several local high schools, and a unit of the American Cancer Society in a needed area.

Her final effort produced an alcoholism and alcohol abuse education program at Meharry. This program teaches students, residents, faculty, clients, and the general community. Today it continues to grow and could become a model for teaching.

In the days following her death, Meharry held a memorial service for Carlisle. In representing family medicine, I shared the following thoughts:

A Tribute To Sharing

We met Carlisle.

We saw a young girl sharing her musical instrument with a schoolmate who could not yet afford her own.

We saw a scholarly young college student sharing her General Physics notes with a classmate competitor because he needed the course more as a Pre-Med major than she as a Chemistry major.

We met Carlisle.

We saw a bright young research chemist, the first and only such female in a giant Texas petrochemical corporation, sharing her discoveries with her colleagues, although she knew that they did not want her around.

We saw a highly motivated young scientist working at the National Institutes of Dental Health to make fundamental contributions to the knowledge of collagen synthesis and function.

We saw a dedicated educator sharing her creations and innovations with eager young minds; tackling the Boston school system and making a difference!

DEDICATION TO THE MEMORY OF CARLIE MARIE GOODWIN

We met Carlie.

We saw a caring health professional who combined her training in education and public health to bring new energies and clear insights to teaching and learning, to curriculum development, faculty development and community development.

We saw a concerned health professional who sensed the full potential of the community and who thought the family was a necessary part of health care. We saw a promising New Meharrian travelling the highways and by-ways of Tennessee, Alabama, Mississippi, Georgia, carrying the mission of Meharry to countless nurses and doctors as she talked to them about teaching medical students.

We saw a resourceful medical educator building a team to teach about and research the problem of alcohol abuse: a team that started with nothing yet in fifteen months became respected among its peers.

We saw a young woman sharing her energies, her talents, trials, and triumphs, herself, her world with her friends, family, and colleagues.

We met Carlie again this last week, when we found prominently displayed in her home the following writing by an unknown author:

one night i had a dream.....

i dreamed i was walking along the beach with the Lord and across the sky flashed scenes from my life. for each scene, i noticed two sets of footprints in the sand; one belonging to me, the other to the Lord.

when the last scene of my life flashed before us, i looked back at the footprints in the sand. i noticed that many times along the path of my life, there was only one set of footprints. i also noticed that it happened at the very lowest and saddest times in my life. i questioned the Lord about it.

"Lord, You said that once i decided to follow You, You would walk with me all the way, but i have noticed that during the most troublesome times in my life, there is only one set of footprints. i don't understand why, in times when i needed You most, You would leave."

the Lord replied, "My precious child, i would never leave you during your times of trial and suffering. when you saw only one set of footprints, it was then that i carried you!"

We knew Carlie.

--JEA 10-04-82

PREFACE

By Michael Liepman, Raymond Anderson & Joseph Fisher

The Society for Teachers of Family Medicine [R*STFM] Task Force on Substance Abuse (TFSA) was organized and chaired by Raymond C. Anderson to explore the needs of membership in the specialized area of substance abuse and to recommend solutions for them. Under his leadership, and with the support of STFM officers, the TFSA resolved to develop a curriculum monograph addressing the teaching needs of residency programs. With the approval of STFM, financial support for this project was solicited with the help of Joseph V. Fisher, a member of the Health Professions Task Force, and support was offered in the form of two federal contracts, one for preparation of the monograph¹ and the other for free distribution of one copy to each family medicine residency training program in the United States². Additional copies will be available for sale through the STFM.

The authors were selected from among STFM members, Career Teachers in Alcohol and Drug Abuse, recipients of the special substance abuse training funding preference initiative for undergraduate medical student family medicine curriculum development, and selected other topic experts. The editors and authors hope that the monograph will encourage and facilitate family medicine residency training programs to include more effective training in the care of patients and families suffering from chemical dependency. And where undergraduate medical students are also exposed, we hope that the knowledge, skills and attitudes of the residents and faculty will rub off.

Alcoholism and other drug dependencies have reached epidemic proportions in North America and other parts of the world. These are diseases that spare no age, ethnic group, nor social class. The family physician is a key person in early intervention. He or she can prevent unnecessary physical, emotional, or social damage so characteristic of the untreated clinical course of these diseases. Early recognition reflects the preventive medicine mandate of family medicine; but it also provides an improvement in the prognosis for recovery. The philosophical orientation of the family physician--embracing a broad concern for the individual and family within the community--is ideal for considering the addictive diseases as multisystemic biopsychosocial processes. The chemical dependencies represent an excellent demonstration of the power and vitality of the family medicine approach to problem identification and solution.

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² Alcohol, Drug Abuse & Mental Health Administration (ADAMHA)/ National Institute on Alcohol Abuse & Alcoholism (NIAAA) Contract No. 83M050931301D

The general goal of this monograph is to prepare the family medicine educator to provide training experiences that will improve the understanding, attitudes, and clinical skills of residents in dealing with the chemical dependencies within their own practices. In order to do this, it is necessary to supplement the prior training of the educators. This can be done by providing state of the art updates on what is known about the chemical dependencies (a field progressing rapidly in the research laboratory) so that educators can keep pace with learners and remain clinically more skillful as clinician-teachers. Some teachers and residents may wish to learn and do much more than others. These decisions may be spurred by personal interests, as well as by an awareness of the presence or absence of specialized care for this population in the community in which the physician plans to practice. We understand that some family medicine educators are non-physicians, nonclinicians, or specialists. We suggest that these professionals concentrate on those sections which apply to their activities. The STFM Task Force has developed the following specific learning objectives for the readers of this monograph:

1. The teacher should understand the basic physiology, pharmacology, toxicology, and medical/behavioral effects of alcohol and other abusable drugs when these are used in a harmful way by a patient.
2. The teacher should be able to identify, as early as possible, either within his or her own practice, or in that of residents under the supervision of the teacher, those patients or families at high risk of suffering or developing chemical dependencies.
3. The teacher should be able to show the resident how to confront and motivate the patient and family suffering from chemical dependency to seek help and recover.
4. The teacher should be able to show the resident how to provide support for the family whether or not the chemically dependent person recovers.
5. The teacher should be able to demonstrate and supervise safe detoxification procedures in an inpatient or outpatient setting, and be able to manage complications that arise during withdrawal.
6. The teacher should be able to supervise the resident in helping the patient and family to recover and adjust to the recovery.
7. It is not expected that all family physicians will become substance abuse experts, but they should demonstrate minimal skills in recognition and referral to appropriate resources in the community which can supplement clinical limitations. The teacher should train the resident to know his or her own strengths and limitations.
8. The teacher should be able to teach the resident to collaborate with the external treatment resources, including advocacy for the patient and family when dealing with these resources, and likewise advocacy for treatment when dealing with the patient and family. The teacher should be able to demonstrate the utility of medical expertise and familiarity with the whole family

- and social network of the patient in order to plan, coordinate, and monitor the care being provided by local resources.
9. For faculty who have never experienced clinical success with this patient population, the Task Force hopes this monograph will open new horizons for clinical and teaching success with chemically dependent patients and their families.
 10. The teacher should communicate to residents an awareness of the high risk for chemical dependence that exists among health-care professionals and their families. This should influence any advice that is given to residents about personal and occupational matters. It should also be considered when planning group recreation and celebrations in the residency training program. One may wish to reflect on one's own experiences, past and present, with chemical use and abuse, as well as that of one's family and friends, to discover the origin of one's attitudes towards chemically dependent patients.
 11. The teacher should recognize his or her teaching limitations, and refer the student to those who can supplement their education.

In this monograph, we use the terms CHEMICAL DEPENDENCY or DEPENDENCE to mean the chronic tendency of a person to ingest psychoactive chemicals despite consequences that are damaging in the biopsychosocial spheres of life. These chronic behavioral disorders such as alcoholism, street drug dependence, tobacco dependence, caffeine dependence, and dependence upon prescribed opiates or tranquilizers resemble one another more than they differ. The self-administered substances are DRUGS (meaning psychoactive drugs), which vary in their specific pharmacology and hence in their specific effects on behavior. But the basic process of addiction and the biological and social reactions to it remains generally the same. The terms ALCOHOL or other DRUG ABUSE (or SUBSTANCE ABUSE) and DRUG MISUSE are specific terms designating drug self-administration behavior that leads to problems or increases risk of problems. Substance abuse behavior is found in persons suffering from chemical dependence (not in remission) or in some who experiment with these drugs. Generally, patients who ABUSE substances are thought to do so for recreational or self-medication purposes, with the intent to administer the drugs differently than they have been advised. Drug MISUSE indicates a patient who is being prescribed drugs but uses them inappropriately usually out of ignorance about the proper use of the drugs. Most common in the poorly educated or elderly who must take concurrently several drugs, and in persons who have discontinuous health care with multiple doctors prescribing independently, when they discover the error, there is generally no resistance to correcting it. In the drug DEPENDENT person, drug dependency behaviors include much more than mere ingestion. These behaviors include the concealment of drug use, the generation of excuses for drug use, the procurement of the drug(s), and the facing (or not facing) of the consequences of drug use. The chemical dependencies appear to be chronic disorders which can be

PREFACE

treated and brought into clinical remission by cessation of the substance abuse, but a high lifelong risk of relapse is carried by the recovering patient. Recognizing the chronic nature of the illness, rather than speaking of CURE, professionals skilled in chemical dependency treatment talk of REMISSION; instead of RECIDIVISM, we talk of RELAPSE.

The Task Force recognized that faculty and institutional resources and structures differ from one training program to another. Where possible, we have tried to present a variety of strategies to meet the various objectives. Our goal is not to dictate what must be taught or how it is to be taught. Rather, we have tried to provide interested teachers with a guide to facilitate quick access to resources and suggestions from those who have been teaching the desired clinical skills in family medicine or similar settings. We invite the reader to creatively design other ways to teach these lessons. The Task Force also recognizes that any educational program must be supported by the residency faculty. For this reason, we included a special section on strategies for faculty development and curriculum change. Although the major focus of this monograph is residency-level training, many sections may also be useful in training undergraduate medical students and trainees of other health care disciplines. When a generic approach seems appropriate, suggestions are geared towards the STUDENT rather than the RESIDENT. Because the field of addictionology has not become standard in all medical schools, there is considerable variations in the backgrounds of residents coming from different schools. Consequently, it will be necessary for the teacher to gauge the level of baseline knowledge, skills and attitudes of each resident before deciding at what level to direct teaching.

A critical factor in meeting any of the behavioral objectives of skills training in chemical dependence is to have a positive impact on the attitudes of the learner. This is most successfully accomplished by having exposure to both individuals who are recovering from chemical dependence, and to clinicians and teachers who are enthusiastic about helping these patients. Studies have shown that medical students develop progressively more negative attitudes towards alcoholic patients as the students' training progresses. But these attitudes can be shifted positively when students are exposed to an educational program on chemical dependencies. Sometimes attitudes only change after behavior changes--for instance, if a skeptical resident somehow participated in helping a "hopeless alcoholic" to recover, the optimism of the resident is likely to increase dramatically [J*Pursch, 1978].

REFERENCES

Unfortunately, it was not until the late 1970s that the Index Medicus and the Citation Index began to carry references to articles published in the substance abuse specialty journals, so many good articles are difficult to find. While the bibliographic and resource list is not complete, it is hoped that the reader

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will have an adequate supply of references to explore most issues that arise in the teaching setting. The citations include many of the standard textbooks and reference works and most of the journals that address substance abuse issues, so those who wish to explore issues in greater depth may probe these first.

The reference section for the entire monograph is at the end in four sections. Audiovisuals, books, journals and resources are cited in the text simply in the following format: [A*name] for an audiovisual, [B*author, date] for a book or chapter, [J*author, date] for a journal article, and [R*name] for a resource.

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⁵ on a Prism 132 printer with sprint mode [R*Integral Data Systems].

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CHAPTER 1: PHARMACOLOGY OF COMMONLY ABUSED DRUGS

By E. P. Schoener, E. D. Nelson, R. E. Parker, & L. T. Sigell

OVERALL OBJECTIVES

Knowledge of the fundamental pharmacodynamic and pharmacokinetic properties of abused drugs will permit the family medicine resident to correctly diagnose and treat patients who exhibit signs of drug intoxication, overdose, interaction, tolerance and dependence (withdrawal). For each class of drugs, the resident should understand and be able to relate the following to diagnosis and management of substance abuse, misuse or dependency and related disorders or complications:

1. Chemical description of substance or its components;
2. Typical pattern(s) of use;
3. Absorption and distribution throughout the body, considering different routes of administration where appropriate;
4. Metabolism of active and inactive metabolites;
5. Elimination pathways and kinetics;
6. Mechanism(s) of action at tissue, cellular, and molecular levels;
7. Mechanisms of tolerance and dependence (if applicable to that drug);
8. Acute and chronic effects on healthy and disordered body systems and functions; and
9. Interaction of effects with those of other common drugs.

COMMENTS : Introduction

The use of mind-altering substances dates from antiquity. Our ancestors knew little of pharmacology, yet their shamans provided them with psychogenic experiences through an extensive pharmacopoeia of root, seed pod, and fermentation products. Empirically determined doses of natural products were recommended for desired religious and mystical revelations.

During the many years since then, our understanding and sophistication about mood-altering agents have grown to the extent that we can now describe molecular mechanisms of action in great detail for many of these substances. Yet, with all this knowledge at hand, we must appreciate how much more remains unearthed about drug action and brain chemistry. Perhaps in the not too distant future, motivational processes underlying substance abuse will be described in similar detail.

Information about this topic may best be presented for each class of drugs:

CHAPTER 1: PHARMACOLOGY OF COMMONLY ABUSED DRUGS

1. depressants, including ethanol, sedative-hypnotics, general anesthetics and tranquilizers;
2. opioids;
3. psychomotor stimulants;
4. hallucinogens, including phencyclidine and marijuana; and
5. inhalants and solvents.

Following this will be a section dealing specifically with drug interactions.

LEARNING OBJECTIVES: Pharmacology

The learner should be able to (1) list and give examples for the four primary classes of abused drugs, and (2) list eight areas of consideration essential to the understanding of drug-abuse pharmacology.

TEACHING HINTS: Pharmacology of abused drugs

Didactic forms of instruction are most appropriate for this material. These may include the lecture format, the reading/tutorial seminar approach, or, if suitably modified, a self-instructional program. For the latter, it would be necessary to develop several series of self-evaluation questions to accompany the text, and provide the learner with adequate feedback regarding comprehension and retention of the information.

EVALUATION: Pharmacology of abused drugs

This subject matter lends itself best to the most fundamental and direct modes of evaluation. Thus, a written examination would be entirely appropriate. However, the author believes that multiple-choice-type items should be avoided, and emphasis be placed on provocative essay questions that require synthetic process and some interpretation of data.

DEPRESSANTS

COMMENTS: Ethanol

In today's rapid-paced world, the chemical depressants have retained their long-standing popularity against mounting challenges by stimulants and hallucinogens. In fact, alcohol has never relinquished its preeminent position on the ever-growing list of recreational substances (excluding caffeine). As one might surmise, the drug-abuse problem of greatest moment in our nation today is that of alcohol.

TABLE 1-1: Ethanol content of standard servings

BEVERAGE	PERCENT ETHANOL	STANDARD DRINK	OUNCES ETHANOL	MILLILITERS ETHANOL	GRAMS ETHANOL
Wine (goblet)	12%	4 oz.	0.48 oz.	14.4 ml.	11.4 gm.
Beer (can)	5%	12 oz.	0.6 oz.	18.0 ml.	14.3 gm.
Liquor (80 pf shot)	40%	1.5 oz.	0.6 oz.	18.0 ml.	14.3 gm.
Liquor (86 pf shot)	43%	1.5 oz.	0.65 oz.	19.5 ml.	15.4 gm.
Liquor (100 pf shot)	50%	1.5 oz.	0.75 oz.	22.5 ml.	17.9 gm.

VOLUME PURCHASES

Wine (liter)	12%	25 oz.	3.0 oz.	90 ml.	71.5 gm.
Beer (6-pack)	5%	72 oz.	3.6 oz.	108 ml.	85.7 gm.
Beer (case)	5%	288 oz.	14.4 oz.	432 ml.	343 gm.
Pint liquor (100 pf)	50%	16 oz.	8.0 oz.	240 ml.	191 gm.
Fifth liquor (80 pf)	40%	25 oz.	10 oz.	300 ml.	238 gm.

MEDICINAL ALCOHOL

Therapeutic use of ethanol is extremely limited. Indications include retardation of uterine contractions during parturition (ethanol has been replaced by newer prostaglandins), neurolysis of sensory nerve tracts from diseased tissue (e.g., inoperable carcinoma), and treatment of methanol poisoning to block the accumulation of formaldehyde. It is also favored as a skin disinfectant and rubefacient. Abuse under these circumstances has never been reported. It is the recreational drinking of alcohol in its various forms that promotes acute and chronic abuse. Conservative estimates place the number of people directly troubled by excessive alcohol intake at approximately 10 million in the United States alone. Untold numbers of others are harmed by the chemical dependency of someone very close to them.

BEVERAGE ALCOHOL

A wide variety of alcoholic beverages are consumed, but an interesting and important observation has been made about use of this extensive selection: regardless of the type of beverage consumed, the "dose" of alcohol consumed in a typical unit serving is essentially constant. A shot of whiskey, a glass of wine, and a bottle of beer contain roughly equivalent amounts of alcohol (see Table 1-1). A corollary of this axiom mandates that severe alcohol related problems can and do result from the excessive use of any alcohol-containing beverage.

CHEMISTRY AND DISTRIBUTION

Ethanol is a hydrophilic molecule which, due to its chemical simplicity and small size, can cross membranes with great facility and can distribute itself widely throughout the body. This perfusion-dependent distribution is necessary to permit the wide-ranging effects of alcohol on so many different body organs and tissues. When taken by mouth, a small amount of alcohol is absorbed in the stomach, but absorption proceeds more rapidly in the small intestine. Peak blood alcohol concentrations are usually achieved at 90 minutes following rapid ingestion, but the rate of absorption is retarded by the presence of food in the stomach, a gradual pattern of ingestion, or high concentrations of ethanol (i.e., >15%) or other sedatives, all of which delay stomach emptying. The ethanol absorbed must normally¹ pass through the liver, carried from intestinal veins into the portal vein. Some may be catabolized in the liver. The excess spills into the hepatic vein and enters the systemic circulation. Once in the plasma, the drug distributes rapidly to well-perfused tissues, less so to fatty depots. Thus, obese persons will show central nervous system (CNS) effects more rapidly and with greater intensity than lean people of equal weight.

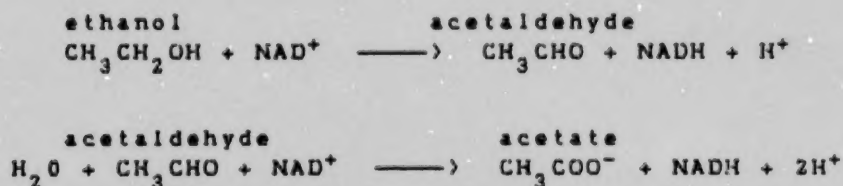


Figure 1-1: Ethanol catabolic pathways

METABOLISM

The catabolism of ethanol [J*Wilkinson, 1980] occurs mostly in the liver. Of the amount consumed, 90-98% is metabolized. The rest is excreted unchanged in the urine, feces, sweat, and even in expired air (which contains approximately 0.05% of the plasma level). The primary catabolic pathway employs the oxidative enzymes alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) to produce acetic acid, which readily combines with coenzyme A and enters the Krebs cycle (aerobic glycolysis). The oxidative reactions catalyzed by ADH and ALDH employ nicotinamide

¹ If there is cirrhosis of the liver and portal vein flow compromise, a portion of absorbed chemicals will directly enter the systemic circulation through dilated esophageal and hemorrhoidal collateral veins (see Chapter 2 for details).

adenine dinucleotide (NAD) as the hydrogen acceptor (see Figure 1-1). Actually, ADH exists in at least two different forms (isozymes) with somewhat differing properties that are responsible for fundamentally different responses to alcohol. For example, "atypical ADH," which is common in Oriental populations, has a catalytic rate four times greater than typical ADH found in most Caucasians [J*Stamatoyannopoulos, Chen & Fukui, 1975]. Thus, acetaldehyde, which is quite toxic, accumulates more rapidly in Orientals than in Caucasians. Newer research has revealed that ALDH also exists in isozymes [J*Hanna, 1978; J*Ijiri, 1974; J*Reed et al., 1976; J*Schuckit & Raynes, 1979; J*Wolff, 1972 & 1973]. Orientals who flush lack the low K_m ALDH₁, and that this reduces tolerance to the behavioral effects^m on nystagmus and body sway [J*Truitt, Rowe & Mehl, 1984]. Ultimately we may find that other personal differences in ethanol effects are also due to this step in drug metabolism.

The primary enzyme ADH is present in a rather limited supply. Because alcohol is consumed in such large molar quantities (relative to most other psychoactive agents), ADH easily becomes saturated with its substrate, and the rate of ethanol metabolism is constant with time and independent of the amount remaining in the body. This is termed "zero-order" kinetics. For most people, the rate is approximately 100 mg/kg/hour, which translates to about 5-10 g/hour or 10-20 mg/dl/hour. Therefore, a typical drink is largely metabolized within 1-2 hours of having been consumed. Tissue distribution of ADH varies, with brain, blood and fetal distribution far lower than liver.

Two additional metabolic pathways exist for ethanol. One of these, called the microsomal ethanol oxidizing system (MEOS), is the same hepatic cytochrome P₄₅₀ enzyme system that is responsible for degradation of many other drugs, including barbiturates. The other pathway, involving the enzyme catalase and a peroxide (H_2O_2) cofactor, results in the formation of acetaldehyde, which may then be channeled through ALDH to the Krebs cycle. While these additional degradative routes do increase the rate of ethanol catabolism to acetaldehyde (to as much as 30 mg/dl/hr), they do not substantially alter the zero-order process of alcohol metabolism, nor do they significantly contribute to the development of tolerance. In this context, it should be noted that tolerance to barbiturates and other depressant drugs in alcohol abusers is due, in part, to the accelerated synthesis of new P₄₅₀ enzymes and the resulting increased metabolic capacity.

MECHANISMS OF PSYCHOACTIVE PROPERTIES

Ethanol has long been considered a general depressant, akin to the volatile anesthetics, although it produces excitation at low doses. Evidence now suggests that we must reevaluate the mechanisms underlying this excitatory potential. For many years, behavioral stimulation resulting from small amounts of alcohol had been attributed to a disinhibitory phenomenon, that is, depression of inhibitory brain centers. This dogma is now being challenged by observations that ethanol can directly enhance

neuronal activity at very low concentrations (orders of magnitude lower than those responsible for CNS depression). Although these findings may eventually lead to new concepts about why people use alcohol, we must not lose sight of the fact that common levels of consumption do result in CNS depression to varying degrees.

Ethanol produces some of its effects through a direct, general action on the excitable cell membrane. This stands in bold contrast to the actions of most psychotropic drugs, which exert their effects through interactions with specific receptors on the membrane. Ethanol, it seems, has the facility to insinuate itself within the lipid portion of the membrane [J*Chin & Goldstein, 1977; J*Chin, Parsons, & Goldstein, 1978]. In so doing, it disturbs the membrane's usual semi-rigid state and destabilizes normal function such that optimal ionic gradients can no longer be maintained. When this occurs in the presynaptic membranes of axon terminals, the release of stored transmitters is enhanced. This may be the mechanism of low-dose excitation. However, when the postsynaptic neuronal membranes are extensively "fluidized" by the same action of alcohol, they can no longer generate and conduct nerve impulses, and so depression results.

Membranes are dynamic structures, characterized by continual turnover of their phospholipid, cholesterol, and protein constituents. It appears that this turnover may underlie development of tolerance and dependence to ethanol. Neuronal membranes seem to respond to the presence of ethanol by selective turnover of components, favoring those with inherently more rigid structures. Thus, membrane concentrations of cholesterol and saturated fatty acids increase, while those of unsaturated fatty acids decrease. With this incorporation of more rigid elements, greater concentrations of ethanol are required to fluidize the membranes and elicit the anticipated pharmacological actions (condition of tolerance). Likewise, normal function is impaired in the absence of the usual titer of ethanol, because the membranes are too rigid to accomplish their role (dependence).

Mechanistic considerations of ethanol pharmacodynamics have enjoyed a lively controversy over the past decade. Discussions have centered upon the significance of tetrahydroisoquinoline (TIQ) alkaloids in alcohol actions [J*Cohen, & Collins, 1970; J*Davis, & Walsh, 1970; B*Deitrich, & Collins, 1977]. The concept that TIQs play a role in chronic and possibly acute alcohol effects is based upon the fact that acetaldehyde condenses with neurotransmitter substances in the brain to form novel, pharmacologically active compounds. Dopamine and serotonin have both been implicated in this process. Apart from the acute pharmacological effects of these TIQs, which in some respects resemble those of the parent biogenic amines, it has been shown that cerebral administration of the agents can generate a preference for alcohol consumption in laboratory rats and monkeys that normally refuse to drink alcohol [J*Meyers, & Melchior, 1977]. Perhaps even more provocative has been the finding that this change of behavior may be irreversible. The implication drawn from these experiments is that in vivo production of TIQ substances may explain the acquisition of alcoholism. One further

observation that has been vigorously contested is that these TIQs possess opiate-like actions. They do indeed express this potential, indicating the possibility of a common mechanism underlying the motivation to use either alcohol or opiates.

EFFECTS OF ETHANOL ON BODY SYSTEMS AND FUNCTIONS

Positional nystagmus occurs as the BAL rises and falls. Ostensibly due to rapid changes of endolymph density in the semi-circular canals, this property may prove clinically useful in the differential diagnosis of ethanol induced CNS depression. It is most visible when the body (head) is in a horizontal position. The eyes slowly drift upward (rising BAL) or downward (falling BAL), and then rapidly jerk back, only to drift again. The vestibular imbalance signaled by nystagmus contributes to the dizziness and nausea experienced in the inebriated state.

Respiratory control, a function of the lower brain stem, is quite sensitive to the depressant action of alcohol. Respiratory drive per se and carbon dioxide responsiveness are both suppressed, and dangerously so at BAL > 300 mg/dl. Cardiovascular regulation is relatively spared by moderate ethanol intake. However, some decrease in peripheral resistance occurs, together with increased heart rate (reflex tachycardia). The alcoholic is also at risk for development of severe hypothermia. Myocardial contractility and cardiac efficiency may also be compromised at elevated levels of consumption, due to cardiac vasoconstriction. This certainly impugns the age-old claim that alcohol is beneficial for heart ailments, and even tempers consideration of newer reports that low-density lipoprotein plasma levels (and therefore plaque formation) are reduced by alcohol.

Gastric secretion and motility are controlled by several factors responsive to the presence or even anticipation of alcohol. Secretion of gastric juice is profoundly influenced by psychic drive and so the very thought of drinking can elicit secretion which is highly acidic. Direct and reflex secretion also occur as a result of gastric mucosal stimulation when alcohol is present in the gut. An acid-rich, enzyme-poor product results from direct stimulation, but at alcohol concentrations below 15%, gut function is not markedly altered. Higher concentrations do inhibit both secretion and peristaltic activity, an unfortunate circumstance, for alcohol itself is irritating to the mucosal tissue. Gastritis is extremely common in cases of regular, excessive use of alcohol.

Organs associated with the gastrointestinal tract, especially the pancreas and liver, are sensitive to alcohol. Pancreatitis and hepatitis are frequent sequelae to chronic alcohol abuse, but both organs exhibit more subtle changes with acute use. Precise pathogenetic mechanisms for the pancreatic inflammatory response to alcohol are unknown. Mechanisms of hepatic injury have been more extensively investigated, revealing that the primary cause here is deranged lipid (and possibly protein) metabolism. Because massive ethanol metabolism results

in large scale accumulation of NADH, lipid synthesis is augmented in a nonspecific, uncontrolled fashion. Compounding this process is the secondary hepatic accumulation of fat, which is mobilized by the lipotropic action of alcohol on peripheral stores. Well-conducted studies have shown that these effects are the direct result of alcohol and not the consequence of the nutritional deficits that often accompany the abuse of alcohol.

Endocrine glands under the control of tropic pituitary hormones show greatly accelerated release, while testosterone synthesis is impeded and its catabolism enhanced. The latter effects are often responsible for the testicular atrophy seen in male alcoholics. Adrenomedullary release of catecholamines is increased and most likely responsible for elevated rates of glycogenolysis and lipolysis. Hyperglycemia and lipidemia are closely associated with acute alcohol intake.

Intoxication

Alcohol is used recreationally to create an altered state of consciousness. Our attention, therefore, naturally focuses on the acute CNS actions of the drug (for chronic effects, see Chapter 2). As a person drinks alcohol and it is absorbed into the body, CNS effects begin to accumulate [J*Wallgren, 1963]. The neurological and behavioral manifestations are more pronounced at a given blood alcohol level (BAL) when the BAL is rising than when it is falling, a reflection of tachyphylaxis or acute tolerance. The rate of rise of the BAL is the best predictor of the degree of neurological dysfunction and the subjective feeling of intoxication. Low doses are associated with elevation of mood and enhanced sociability. Acetaldehyde has been implicated in the euphorogenic properties of alcohol [J*Brown, et al., 1983]. Task performance, however, is not improved. Progressively greater quantities of alcohol depress the mood as well as motor function in a dose-related fashion, as exemplified by the slowing of body movements, slurring of speech, and general disorientation (see Table 1-2). Intermediate doses are characterized by enhanced aggressiveness and risk-taking; alcohol-related accidents, homicides, suicides and child abuse occur at this point. With sustained consumption, perception and judgement decline in parallel fashion. It becomes progressively more difficult for the drinker to select a dominant focus of attention. Electroencephalographic (EEG) records visibly illustrate the neurologic concomitants of these behavioral changes: alpha waves progressively become slower and larger as intoxication proceeds, and a delta pattern is ultimately achieved. Sleep ensues, but is not restful, because rapid eye movement (REM) periods are curtailed [B*Hauri & Proeger, 1983; B*Orr, Altshuler & Stahl, 1982].

TABLE 1-2: Neurologic Manifestations of Blood Alcohol Levels in Non-Tolerant Drinkers

BAL	NEUROLOGIC STATUS
50 mg/dl	mild reduction of inhibitions/feeling of warmth & relaxation/impaired thought & judgment
100 mg/dl	ataxia/impaired sensory discrimination/slow reflexes/exaggerated emotion & behavior/denial of impairment/noisy/morose/legally intoxicated
150 mg/dl	clumsy/belligerent
200 mg/dl	lethargic/emotional outbursts/motoric retardation
300 mg/dl	confusion/stupor
400 mg/dl	disorientation/coma
500 mg/dl	respiratory & vasomotor depression/death

Tolerance & Dependence

A person who uses alcohol to excess regularly, and for a long period of time (e.g., daily consumption of 6-10 drinks for several weeks) will experience the development of tolerance and dependence. The physical and emotional impacts of this state are overwhelming, driving the person to continue drinking in progressively greater quantities. The tolerant person must increase the dose to achieve the desired pharmacologic effect and in the dependent state must continue using the drug regularly or endure the discomfort and danger of an abstinence syndrome. The person with tolerance who experiences euphoria may deny the reality of the neurological impairment which others observe objectively. A rough estimate of the degree of neurological impairment in the non-tolerant individual is shown in Table 1-2. The drinker with tolerance or cross-tolerance will show less impairment than is listed on Table 1-2 at any BAL that he or she has repeatedly experienced in the recent past. The drinker who has other potentiating drugs in the body in addition to the ethanol will be likely to show more impairment than is suggested by Table 1-2. If both conditions apply (i.e., the patient has tolerance and has been taking drugs while drinking), then it is impossible to accurately predict the degree of impairment that would be associated with any particular BAL.

Irritability and craving are psychological signs of impending withdrawal; they drive the afflicted person to seek relief with yet another drink. However, if alcohol is inaccessible and

the BAL falls precipitously, the person may experience an alarming constellation of physical symptoms, including:

1. tremulousness;
2. nausea and vomiting;
3. tachypnea;
4. postural hypotension;
5. labile blood pressure;
6. tachycardia;
7. pyrexia;
8. diaphoresis;
9. hallucinogenesis;
10. disorientation;
11. misinterpretation of sensory stimuli;
12. convulsions.

Although there is a specific time course for the alcohol withdrawal syndrome, (see Chapter 5), the manifestations of withdrawal do not always occur in any definite chronological sequence nor must they all appear in a given episode. Delirium tremens, the unique withdrawal disorder characterized by intense agitation, psychotic delirium, and hyperpyrexia, is truly life-threatening, but fortunately is experienced by less than 5% of those undergoing a controlled, medical withdrawal [P*Sellers & Kalant, 1982].

TEACHING HINTS: Ethanol

Utilize a self-instructional module [B*Schoener, 1981] or readings in standard textbooks [B*Becker, Roe & Scott, 1974; B*Kissin & Begleiter, Vol. 1, 1971; B*Mendelson & Mello, 1979; B*Pattison & Kaufman, 1982; B*Westermeyer, 1976; B*Whitfield, Liepman & Williams, in press].

Present a series of cases to the students that taxes their knowledge of alcohol information. Review a series of emergency ward cases to provide case material. Bring a breathalyzer to class and teach the students how to use one to determine the degree of intoxication of patients. Then discuss the BAL level interpretation in terms of laws about driving intoxicated, tolerance, prediction of withdrawal syndromes, levels of consciousness and neurological impairment.

Have the students do a laboratory experiment where some of them drink alcohol while others monitor their intake, BAL, neurological status, and subjective experience. Then have them monitor the fall in BAL over time. This has been done for several years with undergraduate medical students at Brown University [R*Lewis & Femino]. It is educational and captures student interest.

EVALUATION: Ethanol

Does the student know the interpretation of blood alcohol levels? How rapidly is alcohol absorbed and distributed throughout the body? How quickly is it eliminated? Into which compartments of the body does alcohol travel? How does this influence its effects? What are the chemical properties of ethanol and what are the physiological ramifications thereof? If a patient was brought into the emergency ward in a coma and the BAC was 230 mg/dl, what does this mean in terms of acute management of the case? (Does the student realize that 230 mg% cannot solely cause coma?) If the patient was admitted to the hospital, should withdrawal be anticipated? Should the patient be referred for alcoholism rehabilitation? How would one decide? If the patient is sent home rather than admitted, at what time would the patient be safe to drive again? Can the student explain alcohol tolerance and dependence in mechanistic terms? Can the student describe ETOH effects on CNS function with increasing dosage, and give examples with reference to EEG activity, neurologic, and behavioral signs and symptoms? Does the student understand and can he or she discuss the mechanistic basis for the actions of alcohol on the nervous system? Can the student describe the proposed action of ETOH via synthesis of tetrahydroisoquinoline alkaloids?

Are there any effective antagonists to alcohol? What drugs cause the disulfiram-ethanol reaction (DER) [J*Peachey et al., 1980]? How is it recognized and treated? If the resident encounters the DER, the preceptor can watch how the resident manages the case. How does alcohol interact with other drugs? Which drugs interact? List 8 and describe the mechanisms of interaction.

Does the student know the major consequences of ethanol on body function (see Chapter 2)? Can the student sort out alcohol toxicity from other illnesses? Can the student describe alcohol effects on respiratory, cardiovascular, GI, and endocrine functions? Can the student describe the problems of general anesthesia (for surgery) with an alcoholic patient? Can the student describe conditions leading to and manifestations of the alcohol abstinence syndrome, and list at least 12 systemic manifestations and define delirium tremens in functional terms, and list 3 primary manifestations?

HYPNOSEDATIVES/MINOR TRANQUILIZERS**COMMENTS: Sedatives**

In today's fast-paced, high-tension world, many people resort to depressants (such as barbiturates and benzodiazepines) to relieve the anxiety caused by this lifestyle. Indeed, many physicians, in collusion with their patients, tacitly abet this artificial mood control, rather than recommend the more difficult course of lifestyle change. This is most unfortunate, for it

validates an unhealthy approach to life. It encourages and enables drug-seeking behavior that all too often progresses beyond the professional relationship. Interestingly, women are more apt to suffer from sedativism, that is, the chronic abuse of sedative-tranquilizer drugs, than are men, in a ratio of approximately 3:1 and elderly confined to nursing homes are also at higher risk [B*Brecher, 1972; B*Hofmann & Hofmann, 1975; B*Ray, 1978; J*Scheibel, 1980; B*Smith & Wesson, 1973; B*Thomas, 1979; B*Weiland, 1979; B*Wesson & Smith, 1977].

Sedative abuse is by no means limited to iatrogenic circumstances, though this is the most easily remedied through discretionary prescribing practice. There is much illicit trafficking in drugs of this class, and certain patients will sell valid prescriptions for "downers" much as they would for narcotics. Therapeutic indications for sedatives and minor tranquilizers are distinctly different. Because the former agents induce more profound CNS depression, they are generally reserved for evening or nighttime sedation, whereas the latter are employed for daytime control of anxiety. This dichotomy in practice reflects differences in the mechanism of action between the unique drug types. Actually, because of their more favorable therapeutic index (margin of safety), the hypnotic benzodiazepines are also preferred today for nighttime use.

BARBITURATES AND RELATED AGENTS

LEARNING OBJECTIVES: Barbiturates

The learner should be able to (1) list the groups of barbiturates and discuss their characteristics; (2) describe current theories on the mechanism of sedative drug action; (3) explain the theoretical bases for barbiturate tolerance and dependence; (4) describe barbiturate effects on CNS, cardiovascular, respiratory, and skeletal muscular function; (5) describe phenomena associated with barbiturate poisoning; and (6) explain how other drugs interact with barbiturates, and name several agents that do so interact.

COMMENTS: Barbiturates

Barbiturates are are classified into three groups, [B*Cox et al., 1983] based on their duration of action. Ultra-short-acting barbiturates (e.g., thiopental) have plasma half-lives ($t_{1/2}$) of 3-8 hours; short- to intermediate-acting barbiturates (e.g., secobarbital and pentobarbital) exhibit half-lives of 14-24 hours; and long-acting barbiturates (e.g., phenobarbital) have half-lives of 24-96 hours. The actual duration of sedation may be considerably shorter than these plasma concentration values would suggest, due to a redistribution of such drugs from the CNS to lipid depot sites. This occurs especially with the highly lipid-soluble, ultrashort-acting agents. These drugs also produce their

effects with the shortest latency, highest potency, and briefest duration of action as a result of their more rapid movement across membranes (a lipophilic function). The short-acting drugs are the most highly prized by recreational users of barbiturates, for they seem to evoke the most satisfying experience of euphoria (see Figure 1-2 for the structure of barbituric acid).

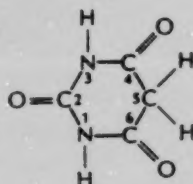


Figure 1-2: Structure of barbituric acid

Catabolism

Barbiturate actions are terminated by mechanisms of both excretion and metabolism. Certain drugs in the group are primarily excreted in their native (active) state and exhibit long duration of action. They are candidates for glomerular filtration because of relatively lower lipid solubility and plasma protein binding. Phenobarbital, a drug employed in prophylaxis of seizure disorders, is one of those drugs dependent on renal mechanisms of elimination. However, the majority of barbiturates are metabolized to inactive products prior to excretion. This process occurs predominantly in the liver, yielding polar compounds more amenable to renal excretion. It is important to realize that the metabolic process is relatively slow, and that with repetitive administration, there is an accumulation of active material that may complicate the pharmacodynamic picture, especially with the addition of other depressant drugs including ethanol.

Mechanism of Action

The specific mechanisms by which barbiturates and related agents produce CNS depression have not been characterized definitively. A number of possible mechanisms have been proposed. Preeminent among them is one that closely follows that described for ethanol. It holds that these depressants have a similar capacity to penetrate the lipid fraction of excitable membranes and thereby cause "fluidization." A distinction appears, however, in that the barbiturates may only accomplish this on the inner cell membrane surfaces (a site of action shared with local anesthetics). These mechanistic considerations explain the more rapid action of the highly lipophilic compounds in the group.

It has also been demonstrated that barbiturates, like ethanol and opiates, restrict the transport of calcium across neuronal membranes. Because calcium is necessary to the process of synaptic neurotransmission, such restrictions may well contribute to the inhibitory actions of barbiturates. However, the

fact that this action is exhibited by a number of different drugs suggests that it may only represent an epiphenomenon. More specific mechanisms of action have been proposed with regard to individual neurotransmitters. Electrophysiological studies have shown that at sedative dosage, barbiturates tend to block excitatory neurotransmitters, most explicitly acetylcholine.

If one single neurotransmitter is implicated in the sedative action of barbiturates, this now appears to be gamma-aminobutyric acid (GABA), a major inhibitory substance in the brain. Although no specific receptor interaction seems to occur between barbiturates and GABA, drugs of this class have been found to improve the intrinsic inhibitory effects of GABA and even stimulate its further synthesis.

Tolerance & Dependence

Tolerance and dependence occur with repeated use of barbiturates and similar depressants as they do for alcohol, but the two phenomena are not mechanistically entwined. Tolerance, or the requirement for increasing amounts of barbiturate to produce the same degree of sedation, is a function of both dispositional and pharmacodynamic factors. That is, the hepatic capacity for metabolism increases (due to enhanced synthesis of mixed-function oxidase enzymes), while tissue sensitivity declines. The pharmacodynamic mechanism may be understood in terms of membrane rigidification, as proposed for ethanol tolerance, but this has not been well established in the laboratory.

Interest in the mechanism of barbiturate dependence has implicated a wide range of phenomena, including changes of specific neurotransmitter synthesis, release, turnover, and binding in the brain, as well as more general alteration of membrane lipids and protein synthesis. For example, it has been found that cholinergic function in the brain, which is generally excitatory, is enhanced in the dependent state, whereas GABA synthesis and binding are concurrently suppressed. As noted earlier, the lipid constituents of excitable membranes exist in a dynamic equilibrium; thus, pharmacologic tolerance and dependence engendered by barbiturates theoretically may result from the incorporation of more cholesterol and saturated fatty acids in neuronal membranes. The observation has been made that protein synthesis is essential for the development of barbiturate tolerance and dependence. Therefore, it is reasonable to propose that new proteins or neuropeptides are implicated in the mechanism. Further research along these lines is both necessary and warranted.

Sedative dependence that is inadequately supported will lead to a depressant-type abstinence syndrome characterized by anxiety, insomnia, increased REM sleep, tremulousness, skeletal muscle weakness, delirium, and in severe cases, seizures of the grand mal type. This is not unlike the syndrome experienced with ethanol withdrawal, and differs primarily in duration and time course. In fact, cross-dependence and cross-tolerance exist for

these substances. If the barbiturate-dependent person cannot obtain the drug of choice, then alcohol may be used to forestall signs of withdrawal.

Pharmacological Actions

Most of the pharmacologic and toxicologic effects of barbiturates are referable to the CNS. General excitability and mood state are altered in a dose-related fashion, with small doses producing euphoria and larger ones resulting in depression. The duration of depression is variable. Some patients experience persistent, often disconcerting, clouding of sensibility and mentation for up to 24 hours after a usual therapeutic dose. They describe their cognitive state as hazy and report feeling hung over. Both the peripheral nervous system and the CNS are affected by barbiturates. Clinically significant depression of sympathetic ganglionic transmission occurs, and this results in hypotension. Cholinergic transmission at the neuromuscular junction is altered bimodally. Early, enhanced excitability manifested as muscle fasciculation rapidly gives way to more generalized depression that ends with skeletal muscle blocking drugs that are used in surgery (e.g., d-tubocurarine or gallamine). Smaller doses of these drugs would be required in the barbiturate-depressed patient.

Respiration is diminished before any direct drug action on the intercostal and diaphragmatic musculature is apparent. Both chemical respiratory drive and the inherent neurogenic process are inhibited. Loss of hypercapnic drive precedes that of hypoxia. Unlike the effects of opiates, which follow the description to this point, the barbiturates do not interfere with cough and sneeze reflexes, and, in fact, may induce laryngospasm.

Cardiovascular changes concomitant with barbiturate administration are seldom remarkable. Hypotension may occur as a result of diminished sympathetic tone. Myocardial depression and decreased cardiac output are also possible, but these effects are only of major concern in cases of severe barbiturate intoxication. They may be effectively reversed with beta-adrenergic agents and/or cardiac glycosides.

Due to the reduced blood pressure and concurrent facilitation of antidiuretic hormone release, renal activity is suppressed by high doses of the barbiturates. Clearance of active drugs is impaired and so the duration of action is prolonged--an unfortunate "Catch-22" situation. In addition to urogenital inhibition, barbiturates may decrease motility and secretion in the gastrointestinal tract. These systemic manifestations are of most immediate consequence when large quantities of the drugs are consumed.

Rarely, barbiturates elicit paradoxical behavioral excitation at doses that are normally depressant. This is more apt to occur with elderly patients or young children than with healthy young adults. Another seemingly paradoxical response to barbiturates is the production of pain in some patients. Usually

expressed as joint or muscle aches, such discomfort may persist for several days after the drug is discontinued.

Severe barbiturate intoxication is potentially lethal due to respiratory and cardiovascular depression. Neurological signs of central depression generally correspond to the quantity of the drug consumed; reflexes provide a ready index of the extent of the poisoning. Coma is not uncommon and, depending upon the dose, respiratory activity is impaired in various ways. For example, one overdose patient may exhibit polypnea, whereas another may exhibit severely diminished frequency, or even Cheyne-Stokes respiration. In any case, gas exchange is compromised, with a fall in PO_2 and a rise of PCO_2 . Blood pressure declines for a number of reasons, for example, direct inhibition of medullary vasomotor centers, ganglionic blockade, and myocardial depression, and may result in shock. Pulmonary edema and renal failure are likely to ensue at this stage of depression. Under such conditions, measures aimed at reducing the amount of barbiturate in the body are warranted (e.g., lavage, emesis, dialysis, or hemoperfusion), along with support of vital functions.

TEACHING HINTS: Barbiturates

Have the students read about the barbiturates, their pharmacology, their use and abuse [B*Brecher, 1972; B*Cohen, 1981; B*Cox et al., 1983; B*Hofmann & Hofmann, 1975; B*Ray, 1978; B*Smith & Wesson, 1973; B*Schoener, 1981; B*Wesson & Smith, 1977; B*Westermeyer, 1976; B*Wilford, 1981]. Have them give reports to one another about topics of their choice such as detoxification, drug interactions, patterns of abuse, or rational prescribing practices. Have them debate the issue of whether they ought to be used for long-term treatment of epilepsy or insomnia.

Have them watch films and contemplate or discuss dealing with drug abusing patients when they are requesting drugs or acutely intoxicated or withdrawing [A*Born with a habit; A*Doctor, you've been lied to; A*Neonatal abstinence syndrome; A*Treatment of acute drug overdose].

EVALUATION: Barbiturates

Does the resident understand the cross-tolerance and cross-dependence concepts? What would be the effect if a hospitalized patient who had been taking an oral barbiturate as an outpatient could not take oral medication? How would the resident manage the patient to avert oversedation or a withdrawal syndrome? How would the resident manage a barbiturate overdose? What if the patient had compromise of renal function and the drug was phenobarbital?

BENZODIAZEPINES

LEARNING OBJECTIVES: Benzodiazepines

The learner should be able to (1) list at least 5 currently available benzodiazepines; (2) discuss similarities and differences among benzodiazepines, for example, in their distribution, metabolism, and elimination; (3) describe the proposed mechanism of benzodiazepine action that utilizes unique receptors and GABA transmission; and (4) list at least 6 therapeutic actions of benzodiazepines.

COMMENTS: Benzodiazepines

The advent of benzodiazepines more than 20 years ago was both a blessing and a curse. A truly effective group of tranquilizing drugs, their inherent potential for misuse and abuse went unnoticed for many years. Only in retrospect have we come to realize that all problems expressed through human anxiety cannot be appeased with benzodiazepines, and that these agents, like their relatives (the barbiturates), do engender an insidious form of dependence. With this realization, the medical profession now employs benzodiazepines with far greater caution and restraint.

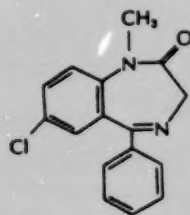


Figure 1-3: Structure of diazepam

Because of their clinical efficacy, thousands of benzodiazepines, such as diazepam (see Figure 1-3) have been synthesized. At least eleven are currently available for therapeutic use in the US (see Table 1-3). Whereas most of these agents are absorbed slowly and distributed over a period of hours, diazepam is rapidly absorbed. This property is most likely responsible for its rapid access to the brain and the euphoriant quality desired by the abusing patient. Most of the benzodiazepines are also highly bound to plasma proteins (85-95%), a fact that results in relatively long elimination times (half-lives) and, in cases of overdose, complicates drug removal by dialysis.

TABLE 1-3: FACTS ABOUT BENZODIAZEPINES

GENERIC NAME	TRADE NAME	FACTOR ^b	HALFLIFE ^a (hours)	DOSES SOLD (mg)	ROUTES SOLD
alprazolam	Xanax	20.0	12-15	0.25/0.5/1	po
chlordiazepoxide	Librium	0.2	8-28	5/10/25	po
	Injectable	?	?	100	iv/im ^c
clonazepam	Clonopin	5.0	18-50	0.5/1/2	po
clorazepate slow release	Tranxene	1.33	30-200	3.75/7.5/15	po
	Tranxene-SD			11.25/22.5	po
diazepam	Valium	1.0	26-53	2/5/10	po
	Injectable			10	iv/im ^c
flurazepam	Dalmane	0.33	>96	15/30	po
halazepam	Paxipam	0.25	50-100	20/40	po
lorazepam	Ativan	5.0	10-20 ^d	0.5/1/2	po
	Injectable		16 ^d	4/8/20/40	iv/im ^c
oxazepam	Serax	0.17	5-15 ^d	10/15/30	po
prazepam	Centrax	?	30-200	5/10/20	po
temazepam	Restoril	0.33	10 ^d	15/30	po
triazolam	Halcion	10.0	1.7-3	0.25/0.5	po

^a Half-life in hours assuming no interacting drugs and normal liver and renal function.

^b To calculate the "dosage equivalent" to the drug in mg. of diazepam, multiply conversion factor by dosage of drug (e.g., 0.5 mg. of triazolam $\times$ 10 = 5 mg. of diazepam).

^c CAUTION: Intramuscular benzodiazepines are erratically absorbed and probably should never be given by the im route if repeated doses are anticipated.

^d Not metabolized in the liver, hence no dosage adjustment necessary in patients with liver dysfunction [J*Gorelick, 1983].

Metabolism is a key point of departure for some of these drugs. For example, clorazepate is essentially inert in its native form. It is converted to its active state in the gastric environment. On the other hand, metabolism of the primary agents by oxidation (N-dealkylation) in most cases results in active products, a factor that prolongs their duration of action. Oxazepam and lorazepam are unique and different in this regard, for they are immediately inactivated by hepatic conjugation with glucuronide, and are rapidly excreted in the urine. Renal excretion is the primary route of elimination for these drugs. However, it has been noted that active as well as metabolic products undergo biliary excretion, leading to enterohepatic recirculation and yet further extension of the sedative action. Clearly, several factors contribute to a complex pharmacokinetic pattern for benzodiazepines. Repetitive administration of large doses can result in the accumulation of dangerously high levels, and these require an exceedingly long time to be cleared.

Benzodiazepines appear to exert their essential influence in the CNS (lower brainstem level), but they do not interfere with autonomic activity at usual therapeutic dosage. Polysynaptic pathways are more sensitive than monosynaptic ones, and we now believe that the mechanism of action is associated with GABA-mediated transmission. Simply stated, benzodiazepines enhance the action of GABA, an endogenous neuroinhibitory substance, for drug action is dependent upon the integrity of GABA systems. It has been proposed that a specific benzodiazepine receptor exists in close proximity to the GABA receptor, and that activation of the former enables function of the latter. Pharmacologic evidence for this hypothesis is rather convincing and interest in possible endogenous ligands for benzodiazepine receptors is keen. Several have been suggested, for example, adenosine, inosine, hypoxanthine, and nicotinamide; however, none fulfills the necessary criteria of specificity and sensitivity. The implications of a benzodiazepine or antianxiety receptor and ligand are far-reaching, and might affect our concept of motivation for abuse of depressant drugs. The possibility that anxious behavior may reflect an inadequate degree of "benzodiazepine receptor" activity is most intriguing.

Tolerance & Dependence

Tolerance and dependence occur with repeated administration of benzodiazepines, especially when high doses are used for prolonged periods (as they are in substance abuse situations). Tolerance appears to be more a pharmacodynamic than a dispositional phenomenon, that is, the increasing drug requirement is due to "down-regulation" of specific receptor function, rather than an accelerated drug catabolism. As yet, explicit mechanisms have not been determined at the molecular level. In view of the different mechanism of actions of the different CNS depressants, it is interesting that benzodiazepines are cross-tolerant and cross-dependent with barbiturates, other sedative-hypnotics, and ethanol.

Clinical Uses

Family physicians and osteopaths wrote 34 million prescriptions (for 5 million patients) for benzodiazepines in 1978 [J*Hollister, et al., 1980]. Benzodiazepines are employed for a variety of purposes in addition to the relief of anxiety. They are used to sedate, to induce pre-anesthetic relaxation and amnesia, for analgesia, for ethanol detoxification, to induce muscle relaxation in spasm syndromes, and as an anticonvulsant. All of these uses rely on the primary CNS effects. Other systemic actions are generally of minor significance, except in toxic dosage, when both respiratory and cardiovascular functions may be depressed. The sedative-hypnotic application can be viewed as a high-dose extension of the lower-dose anxiolytic or tranquilizer effect (i.e., increasing the dose yields greater CNS depression). It has been suggested, however, that the unique antianxiety property of benzodiazepines is more specific in mechanism than CNS depression. It is this distinction that would clearly separate this class of drugs from the barbiturates and other hypnotics. Similar mechanistic considerations arise in the context of benzodiazepine anticonvulsant action, for we now believe that these agents do not directly suppress the epileptic focus, but rather prevent its subcortical propagation. Consistent with the notion of a unique and independent mode of action, tolerance develops to this anticonvulsant property more rapidly than it does for the sedative action.

Use of benzodiazepines as pre-anesthetic medication is predicated on their tranquilizing and depressant effects. Indeed, for some office procedures (e.g., endoscopy), the use of diazepam precludes the need for any general anesthetic. Minor surgery may be possible with benzodiazepines, and it has only recently been appreciated that these drugs contribute a significant measure of analgesia independently of tranquilizing and euphoriant actions. It appears that the analgesia may be both endorphin-dependent and -independent. Skeletal musculature exhibits a selective relaxation with benzodiazepines. These agents are employed quite effectively in spastic conditions, and even for the relief of muscle tension headaches. The action results from specific depression of reticular formation mechanisms that serve to facilitate motor tone at the spinal-cord level.

Detoxification of the alcoholic or sedative dependent patient is conducted most safely and comfortably by empirical sedation by a substitute sedative followed by sequential, progressive drug withdrawal. This procedure seems to avoid or ameliorate the most dire consequences of abstinence (e.g., seizure, hyperthermia, or delirium). For the sake of convenience and control, benzodiazepines (sometimes phenobarbital) are substituted for alcohol on a declining dose regimen, which terminates in 3-5 days. Use of benzodiazepines in lieu of ethanol itself is possible because of the cross-dependency developed by the two. Furthermore, it is now abundantly clear that the chronic, often escalating use of benzodiazepines themselves leads to a disabling dependency. The resulting withdrawal syndrome closely resembles that of alcohol and sedatives, but is slower in

onset. Therefore, in cases of mixed dependency (e.g., alcohol and diazepam), two peaks may be anticipated—one at approximately 36 hours, and the second at 7-10 days after cessation.

In treatment of psychiatric disorders such as acute phen-cyclidine toxicity, acute anxiety reactions, night terrors and somnambulism, obscessive-compulsive disorders, and phobias, the benzodiazepines may have a clinical role [J*Cohen, 1983]. However, caution must be exercised when prescribing these drugs chronically in the absence of proper care by psychiatric experts on the specific disorders, because many of them can be better treated with another modality.

Side Effects

Negative consequences associated with benzodiazepines generally follow their intended actions. For instance, withdrawal seizures are associated with their anticonvulsant effects and rapid development of tolerance and dependence. The amnestic effects so useful in office surgery may produce subtle attention and memory problems which are disabling, and may be mistaken for senility or an affective disorder by the patient, family or unwary physician. The soporific effects of the benzodiazepines may backfire by producing abnormal sleep (deficient in REM phase) which interferes with emotional rest. As tolerance develops, or if the patient reduces the dosage, the baseline insomnia returns transiently, which may convince patient, family and unwary physician that the condition is chronic (most patients and some doctors find it hard to believe that the insomnia would last so long after discontinuing the benzodiazepine because they do not realize the duration of time it takes for the body to fully eliminate the benzodiazepines).

The same problems occur with the anxiolytic effects becoming unmasked as tolerance develops or dosage is reduced. Patients with underlying anxiety disorders such as phobias will note unsuspected return of the symptoms when the dosage is reduced or tolerance develops. A patient who is relying on a benzodiazepine for control of chronic anxiety may appear quite stable and well adjusted while on the medication, even to the extent that the patient and therapist may no longer see the need for psychotherapy. It is believed that pharmacologic reduction of anxiety interferes with normal maturation processes and psychotherapeutic treatment for adjustment reactions such as grief, phobias, and other non-psychotic psychiatric disorders by blocking the normal danger signals of life and warnings of the need to make changes in behavior [J*Lipman, 1980; B*Lipman, 1983]. However, the benzodiazepines can be helpful acutely to stabilize a patient with severe anxiety or whose anxiety may cause adverse complications of other disorders (e.g., acute myocardial infarction, asthma, colitis, peptic ulcer disease). The benzodiazepines may worsen affective depression and may increase the risk of suicidal success through lessening of anxiety over risk-taking behavior. Animal studies have shown that the benzodiazepines block inhibitions that have been taught by

punishment, hence violence, sexual abuse, and other antisocial behaviors may be more likely in some patients while on chronic benzodiazepines.

TEACHING HINTS: Benzodiazepines

Have the students read about the benzodiazepines [B*Brecher, 1972; J*Cohen, 1983; B*Cohen, 1981; B*Cox et al., 1983; E*Hofmann & Hofmann, 1975; B*Ray, 1978; B*Schoener, 1981; B*Smith & Wesson, 1973; B*Wesson & Smith, 1977; B*Westermeyer, 1976]. Have them present topics to one another such as the uses and misuses of benzodiazepines. Or have them debate whether refills should be limited. Have them watch a film [A*Born with a habit; A*Doctor, you've been lied to; A*Neonatal abstinence syndrome; A*Substance abuse and the physician; A*Treatment of acute drug overdose; A*Valium] before this discussion.

EVALUATION: Benzodiazepines

Monitor the prescribing practices of the resident: How many of the patients are being treated with benzodiazepines? What are the indications? Has the resident documented in the record the reasons for choosing the benzodiazepine? How long does the resident intend the patient to use the drug? Has the resident told the patient that there will be a limit on refills? That the drug is habit-forming? Has the resident checked whether the patient is taking any other sedatives or drugs which might interact?

OPIOIDS

LEARNING OBJECTIVES: Opioids

The learner should be able to (1) name naturally occurring, semisynthetic, and synthetic opiates; (2) discuss opiate receptors; (3) describe similarities and differences among opiates, for example, their absorption, distribution, and elimination; (4) understand the concept of endorphins; (5) describe opiate tolerance and dependence in mechanistic terms; (6) describe the actions of morphine on the CNS; (7) describe the mechanism of opiate analgesia; (8) identify the most toxic effect of opiates; (9) list 4 systemic actions of opiates; (10) describe the opiate withdrawal syndrome; and (11) identify the adverse consequences of opiate overdose, and opiate interaction with other drugs.

COMMENTS: Opioids

From its inception more than a century ago, opioid (or narcotic) abuse has attracted considerable attention in this country. Problems first arose from the wide-scale use of opium

and morphine for injuries sustained in the US Civil War. Subsequently, opium and morphine were sold freely as components of patent medicines [B*Eddy, 1973; B*Ray, 1978]. In 1981, analgesics accounted for 110 million prescriptions in the US of which 65 million were for codeine and 20 million were for propoxyphene, and combined analgesics cornered 8% of the pharmaceutical market [J*Baum et al., 1984].

Recreational use of opioids was virtually unknown until the twentieth century. In 1897, the German pharmaceutical manufacturer Bayer introduced a morphine derivative, which they claimed to be superior as an analgesic, but devoid of the detrimental addictive property of the parent compound, allegedly because it is inactivated, if taken orally, in the stomach. The drug was heroin (diacetylmorphine) and its subsequent history of abuse is legend.

Recognition of the growing opioid abuse problem encouraged governmental intervention, and in 1914, the first comprehensive controlled substance-abuse legislation (Harrison Act) was enacted [B*Eddy, 1973]. Those who had become addicted to opioids were no longer able to obtain their supply from pharmacists or doctors because it was decided in the courts that addiction to opioids was not a disease, hence prescribing opioids to such people was unlawful. Since that time, the procurement, sale, and use of heroin and related drugs for other than medicinal purposes has been proscribed by law. And suppliers have sprung up on the street who not only provide solace for the disenfranchised addicted person who cannot locate a physician source of "ethical" analgesia, but they also have continued to recruit new users to recreational use of opioids. Despite vigorous law enforcement efforts, opioid-abuse problems persist.

Typical of all psychoactive substances, the popularity of heroin has waxed and waned over the years. Today, we are witnessing an upsurge in its use that crosses socioeconomic boundaries seldom bridged in years past. It is no longer uncommon to find middle-class, mainstream people hopelessly entangled with opioids. It has been estimated that approximately 2.6 million Americans regularly use narcotics today for reasons other than pain relief. However, not all of these people are necessarily addicted. They may use the drugs on an irregular basis and so never really develop the characteristic constellation of tolerance, physical dependence, and psychological dependence normally attributed to opioid addiction. There are also reports that some persons use heroin in moderation (i.e., "chipping" [J*Zinberg & Jacobson, 1976]) by controlling dosage and frequency to prevent dependence (this must be distinguished from those who use heroin irregularly but in the context of polydrug abuse or while on another drug in a continuous pattern such as methadone or propoxyphene).

Chemistry & Pharmacology

Within this class of drugs we include the naturally occurring substances (morphine and codeine), semisynthetic agents (heroin and hydromorphone), and fully synthetic drugs (meperidine and methadone). All of these possess three structural molecular requirements fundamental to opioid action. These are illustrated in the morphine molecule (see Figure 1-4):

1. a resonant ring (benzene);
2. a centrally located, fully substituted carbon in the D configuration (#13); and
3. a tertiary nitrogen located two carbon atoms away from the quaternary carbon.

The configurational similarities among these compounds suggest that they are all capable of binding to a common pharmacological receptor (membrane recognition site) and are thereby able to initiate the same cellular events that produce narcotic effects. This is indeed the case. The existence of opioid receptors was first proven in 1973. Since that time, we have found that not one, but at least three different types of opioid receptors exist: mu, delta, and kappa. Discrimination among them is based on unique binding properties of various substances in the class.

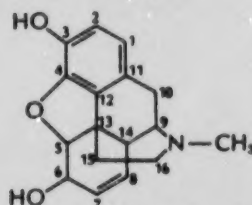


Figure 1-4: Structure of morphine.

Qualitative and quantitative expression of opioid effects differs among substances, partially as a result of differential absorption, protein binding, distribution, and elimination. Opioids are absorbed to various degrees by different routes of administration. For example, upon oral administration, morphine is only one-tenth as effective (bioavailable) as it is by a parenteral route. In contrast, codeine, a less potent drug, has a more favorable degree of oral absorption, and retains two-thirds of its parenteral effect by this route. Both meperidine and methadone have enteral:parenteral ratios of about one-half, and so they are quite effective when taken by mouth. Absorption by the oral route is also efficacious for other commonly employed opioids, including hydromorphone (Dilaudid [R]) and pentazocine (Talwin [R]).

The latency to onset and duration of effects depend upon the particular drug and route of administration employed. By a given route, meperidine and hydromorphone exhibit briefer duration of action than morphine or methadone. On the other hand, when any of the opioids is taken by mouth, a slower onset and longer duration of action can be expected. These phenomena are the result of

pharmacokinetic parameters, notably the extent of protein binding, distribution, elimination, or biotransformation of the drugs. For example, morphine is bound to plasma albumin (ca. 35%), but not to protein in solid tissues. It has a 4-hour duration of action. In comparison, the more lipid soluble agent methadone is highly bound to both plasma and solid tissue proteins (ca. 85%). Therefore, it has a greater capacity to accumulate in the body. With repeated doses, its duration of action can extend well beyond the initial period of 4-5 hours.

Opioids distribute widely in the body. The rate at which they enter the brain is directly dependent upon lipid solubility, and so certain agents act with greater alacrity than others. Heroin exemplifies this point most dramatically. Because its lipid solubility is more than six times greater than that of morphine, it is capable of producing a euphoric, orgasmic "rush," whereas morphine is not. Other lipophilic agents (e.g., methadone) may also be euphorogenic under conditions that enable rapid brain access (high dose and intravenous administration).

Opioids are inactivated by hepatic metabolism, largely by glucuronide conjugation and N-demethylation. However, the primary molecular site of N-demethylation is protected in codeine and heroin, an attribute that is responsible for improved oral efficacy. Meperidine (Demerol) is known to undergo N-demethylation in the liver. This process terminates its pharmacodynamic as an analgesic, but the metabolite (normeperidine) has a potential convulsant action. Thus, it is possible to observe seizures in a patient who has orally consumed a large quantity of meperidine. Both metabolites and active opioid compounds are cleared by the kidneys (>90%) and in the bile (<10%). Because they are charged basic compounds, it is possible somewhat to accelerate the rate of excretion by acidification of the urine in cases of gross intoxication.

Opioids produce various pharmacological effects by virtue of specific receptor binding and activation, as previously noted. Upon recognition of this fact in 1973, a feverish race to identify an endogenous ligand was initiated, predicated on the notion that opioid receptors subserve a normal physiological function and do not exist solely for pharmacologic ends. In two short years, Kosterlitz and Hughes [J*1975] presented the world with a pair of pentapeptide substances isolated from brain tissue that did indeed bind to and activate opioid receptors as well as the opioids. They called these small compounds enkephalins (Enk) and demonstrated that the two were alike in every detail except for the terminal amino acid. The sequences of these enkephalin peptides are: Tyr-Gly-Gly-Phe-(Meth/Leu). Shortly after this revelation, it was found that other similar substances were present in brain tissue that would activate opioid receptors and that these contained the Enk amino acid sequence within them. Chief among these is beta-endorphin, a 31-amino-acid peptide that is synthesized as part of a larger protein (pro-opiomelanocortin), which also contains ACTH, beta-MSH, and possibly other biologically active neurohumoral agents. The term endorphin has been adopted as generic for the entire group of opioid substances

produced in the body. Recently, another seventeen amino acid peptide containing Leu-Enk was isolated from the pituitary gland. It is called dynorphin and may operate as a hormone.

With this proliferation of opioids, and the observation that each possesses a unique distribution, it has been proposed that specific ligand-receptor pairs exist. A truly systematic correlation has not yet been achieved, but it is recognized that certain opioids are more effective on particular receptor types than others (e.g., enkephalin on delta receptors). Opioid drugs (like morphine) seem to have more generalized effects than the endorphins.

Opioid receptors and opioids are found in neuronal and endocrine tissues. When studied physiologically, endorphins generally depress the activity of excitable tissues. They do so apparently by controlling the neuronal input to the end organ, as well as through direct action upon it. Thus, enkephalins, which are synthesized and released by unique neuronal elements of the brain, exert a presynaptic inhibition of neurotransmission, as well as postsynaptic depression of excitability. In this way they perform a neuromodulatory rather than primary transmitter function. The presynaptic mechanism is well documented for systems that employ acetylcholine, norepinephrine, dopamine, and substance P (the pain fiber transmitter) as primary neurotransmitters.

Pharmacologic Actions

The most prominent opioid actions occur in the neurological, nociceptive (pain sensory), respiratory, and gastrointestinal systems. As implied by the very name "morphine" (from Morpheus, Greek god of dreams), opioids are soporific when administered in sufficiently high dosages. However, before sedation is evident, a person will often experience a mood change. Most people, especially pain patients, describe this feeling in positive terms (as a sense of well-being), but some find that it is dysphoric and disconcerting.

Opioids are not general CNS depressants. Although they induce sleep with appropriate EEG wave form shifts, seizure patterns have been observed in EEG records, and frank tonic-clonic convulsions are possible. This is especially true with high doses of meperidine and codeine. It has even been suggested that endorphin dysfunction may be implicated in the etiology of epilepsy. In a practical sense, this proconvulsant activity of opioids must be considered when prescribing other agents that also lower the seizure threshold (e.g., lidocaine and phenothiazines).

Clinical Effects

The analgesia produced by opioids is a selective, specific action [B*Anderson, 1982; B*Olson et al., 1980; J*Terenius, 1981]. Senses other than pain need not be dulled. However, pain

relief may only be complete at dose levels that incur a soporific effect. Dull, continuous pain is most amenable to opioid analgesia; both sensory and reactive components of the pain experience are affected. Pain patients often report that they remain aware of their pain and yet no longer find it discomforting. The differential effect is not wholly understood, but we do know that enkephalins modulate transmission of nociceptive information in the spinal cord as well as in the brainstem, and in higher centers where the processing of pain information takes place.

The treatment of pain has been a difficult problem for modern medical practice because of fear of inducing opioid dependence as a side-effect [B*Brena, 1982b; J*McGivney & Crooks, 1984]. For acute pain, it has been shown that a brief exposure to opioid drugs induces very few cases of drug dependence, and even fewer cases of drug addiction. However, the need of patients for pain relief by pharmacologic agents tends to be a function of the degree of anxiety the patient has about the pain and the type of schedule on which analgesics are offered [B*Johnson, 1982; B*Melzack & Wall, 1982; B*Morse, 1982; B*Pilling, 1980]. Brena [B*1982b] suggests using a fixed schedule rather than a prn schema for administering opioids so as to prevent the conditioned mental link of pain relief to opioid ingestion. The only major exception to this is when prescribing very long-acting agents such as levorphanol or methadone, where there is some danger of drug accumulation and overdosage with regular schedules, and there is little danger of psychological (i.e., learned) dependence as a consequence of prn usage (because of the delayed onset of action characteristic of the longer acting drugs) [J*McGivney & Crooks, 1984].

Many difficult cases arise when a physician or nurse tries to restrict the dosage or prolong the interval between doses beyond that which is reasonable for the patient. The patient becomes worried about the availability of pain relief; ultimately, the anxiety worsens the subjective pain and promotes an increase in requests for analgesia, which in turn may worry the caregiver about the risk of addiction [J*McGivney & Crooks, 1984]. Acute pain patients tend to do best when fully aware of the type and extent of pain to expect, the time course over which it should abate, and the plan for pain relief. If it typically takes an hour for the nurse to administer an opioid dose from the time of patient request, clearly the patient should be instructed to notify the nurse well in advance of needing the next dose, and the nurse should realize that the early request is not a sign of emerging drug dependence.

For patients with chronic pain from a progressive fatal disorder such as cancer, opioids may provide relief of pain and retention of some degree of function until death approaches [J*Walsh, 1983; J*Walsh & Saunders, 1983]. However, their use should be clinically monitored to avoid excessive dosage to the extent that adverse side-effects ensue; sometimes excessive analgesia will be administered by family or health care professionals to allay their own suffering at watching the patient die.

For chronic painful disorders that are neither progressive nor fatal (such as arthritis, back pain syndromes, headaches [J*Dalessio, 1981; B*Saper & Magee, 1981]), there is little legitimate role for chronic opioid administration. The patient and family might be better off with alternative means for pain relief such as biofeedback, relaxation, hypnosis [B*Chapman, 1982], transdermal electrical nerve stimulation [B*Wolf & Rao, 1982], or acupuncture [B*Brena, 1982a; J*Melsack & Katz, 1984] in addition to adequate measures to correct any reversible pathology that may be causing the pain [J*DeFelice & Sunshine, 1981]. However, in cases of severe pain of a relapsing nature (e.g., sickle-cell disease, porphyria, hemophilic hemarthrosis) where physical dependence has already developed, it is best to maintain the patient on a low dose (10-20 mg twice daily) of methadone to eliminate the emergence of withdrawal symptoms and then use acute doses of a short acting opioid agonist for the acute episodes of pain [J*Nyswander & Dole, 1984]. A greater risk of dependence may derive, in part, from concurrent emotional factors such as depression at the time of opioid administration. A patient receiving narcotic analgesics who finds subliminally that the medication relieves the dysphoria of depression, may continue to report pain to the physician long after the need to treat physical pain has passed. This may lead to the patient becoming trapped in the chronic sick role, invalidism, and hypochondriasis, which in turn may produce polypharmacy and polysurgery [J*Sternbach, 1981].

The opioid effect of greatest concern (with regard to toxicity) is respiratory depression. This occurs to some extent under therapeutic conditions, but is a frequent cause of death in cases of heroin overdose. The depression is due to inhibition of both carbon-dioxide and neurogenic ventilatory drive. Where depression is critical, it can be reversed pharmacologically with the aid of naloxone (Narcan [R]), an opioid antagonist. However, the duration of action of this agent is considerably shorter than that of morphine or heroin and especially oxycodone (Percodan [R]) or methadone; hence, repeated administrations may be required.

A number of other actions are well documented for opioids. Some of these are therapeutically useful, others merely indicative of opioid intoxication. These include nausea and emesis due to activation of the chemoreceptor trigger zone in the brain; decreased peristalsis in the gut and resultant constipation; cough suppression (antitussive action); miosis or "pinpoint pupils"; and hypotension caused by peripheral vasodilation and histamine release. Because endorphins and their receptors are widely distributed in the body, opioids (when administered chronically) also have detrimental effects on sex organs. This is largely the result of inhibited FSH and LH secretion. Sexual dysfunction is a known concomitant of long-term narcotic abuse.

Tolerance & Dependence

When opioids are administered frequently for several days or more, a physical dependence develops, that is, performance of normal physiological activity requires drug presence. The extent of dependence is a direct function of the amount of drug used and the specific frequency. If administration is abruptly terminated, or if a narcotic antagonist (naloxone) is given, an abstinence (withdrawal) syndrome ensues. This is characterized by early restlessness and irritability, followed by an inability to concentrate on anything other than securing more of the drug. Somatic signs and symptoms include perspiration, lacrimation, rhinorrhea, piloerection, intense yawning, loss of appetite and vomiting, abdominal cramps, tremor, joint and muscle pain, weakness, and malaise. Pupil size, formerly miotic, will enlarge to greater than normal dimensions. Blood pressure, heart rate, respiratory frequency, and body temperature all increase. To the uninitiated, the constellation of symptoms might suggest a particularly malevolent viral infection.

These manifestations of withdrawal begin within about 6-12 hours after the last drug (heroin, morphine) dose. Onset is more rapid for meperidine (3 hours), slower for methadone (24-48 hours). Peak intensity occurs at about 36 hours for heroin, at 10 hours for meperidine, and in 3 days for methadone. Duration of this syndrome is approximately 5-6 weeks for heroin, 1 week for meperidine, and 6-7 weeks for methadone. However, after this obvious withdrawal passes, the patient does not return to a truly normal state for several months. This period of persistent abstinence is referred to as the secondary, chronic, or protracted withdrawal state. For the abusive patient, it represents a time of very high risk for return to drug-taking behavior if the opportunity presents itself.

Dependence engendered by prolonged opioid use can be understood in cellular and molecular terms, much as we comprehend the dependence that develops with steroid hormone administration. The natural response of endorphin systems to the presence of relatively long-acting drugs is a decreased synthesis of new opioid substances. Chronic administration therefore creates a reliance upon the drug for normal function. Simultaneously, the persistent occupation of opioid (endorphin) receptors seems to reduce their responsiveness and promote the synthesis of new receptors. This yields a greater drug requirement for systemic activation, or in other words, a condition of tolerance.

TEACHING HINTS: Opioids

Have the students read about opioids [B*Brecher, 1972; B*Cohen, 1981; J*Fultr & Senay, 1975; B*Hofmann & Hofmann, 1975; B*Ray, 1978; B*Schoener, 1981; B*Schuckit, 1979; B*Verebey, 1982; B*Westermeyer, 1976]. Have them debate the issue of use of opioids in treatment of chronic pain. Have them discuss the treatment of acute pain in opioid dependent patients. Have them debate methadone maintenance versus drug free treatment of heroin

addicts. Have them discuss opioid overdose management and detoxification. Have them discuss how they would manage a methadone maintenance patient who was hospitalized with a surgical abdomen.

Have them watch a film to spark discussion about pain, addiction, withdrawal or brain enkephalins [A*Born with a habit; A*Doctor, you've been lied to; A*Gale is dead; A*Keys of paradise; A*Neonatal abstinence syndrome; A*Substance abuse and the physician; A*Treatment of acute drug overdose].

EVALUATION: Opioids

Have the students explain the role of exogenous opioid drugs upon the brain opioid receptors if given acutely or chronically. How do the various available opioid drugs differ in their actions and side-effects? Test their concern for seizure risk with a case of an epileptic with a cholecystectomy being treated for postoperative pain by Demerol[R]. If this patient was found in a stupor, how would they differentiate a post-ictal state from an opioid overdose? If they suggest administering naloxone, have the students describe the withdrawal syndrome which might ensue? How often would they have to administer naloxone if it was an overdose of codeine? How long if it was oxycodone or methadone?

STIMULANTS

LEARNING OBJECTIVES: Psychomotor Stimulants

The learner should be able to (1) identify "acceptable" and illicit stimulants used in our society; and (2) describe patterns of amphetamine and cocaine use. Concerning amphetamines, the learner should be able to (1) describe the chemistry, absorption, distribution, and elimination of amphetamines; (2) explain the mechanism of action for amphetamines; (3) describe tolerance and dependence exhibited by amphetamines; (4) describe the systemic actions of these drugs; (5) discuss the effects of amphetamines on the CNS; (6) describe the patient with chronic amphetamine intoxication; and (7) explain the interaction of amphetamines with other drugs. Concerning cocaine, the learner should be able to (1) identify the means of cocaine use; (2) discuss possible mechanisms of cocaine action; (3) describe the acute and chronic CNS effects of cocaine; and (4) identify toxicity associated with acute cocaine overdose and chronic high-dose consumption.

COMMENTS: Psychomotor Stimulants

The most widely used mood-altering substances in American society today are the stimulants. Although the spotlight has focused on abuse of amphetamine and, more recently, on the growing abuse of cocaine, we cannot lose sight of the fact that as a

nation we consume vast quantities of caffeine and nicotine, substances that are by no means innocuous. The use of caffeine in beverage form, and of nicotine through tobacco, is generally condoned and seldom problematic. Health-conscious people are nonetheless taking stock of the benefits and risks entailed in their use, with the evident prospect of curtailing it. The establishment of numerous "smoke-ender" clinics attests to this fact.

The more potent stimulants, amphetamine and cocaine, have enjoyed greater popularity of late, but the patterns of use vary considerably. Much use is actually confined to the occasional, purposeful pursuit of enhanced energy and acumen, for example, studying for exams, driving through the night, or playing competitive sports. However, at the other end of the spectrum, we find "speed freaks" and "coke heads" whose whole lives focus on stimulant dependency.

Amphetamines and similar drugs are legally manufactured for therapeutic purposes, which should be regarded as rather limited. Narcolepsy and childhood hyperkinesis are rational indications for amphetamine trial, but most prescription amphetamine use seems to derive from the ill-advised, chronic treatment of obesity. At least 5 million male US citizens in their 20s² have used major stimulants at least once [B*O'Donnell et al., 1976]. More than 5 million prescriptions are written for amphetamines each year. A good deal of the legal drug seems to be diverted into the illicit market by manipulative patients, "scrip doctors," unethical pharmacists operating in collusion with them, and theft.

Cocaine has been used for centuries in its native environment of the Andes (Peru and Bolivia) without any apparent detriment. This is most likely due to its pattern of use, which more closely resembles our consumption of coffee than our use of amphetamines. The leaves are chewed and the substance is naturally extracted from the juice. In this form it is not regarded as dangerous and it is taken in doses that are frankly not euphorogenic. Cocaine is currently used in a very different manner in urban Lima and in the United States; here the attempt is made to maximize the acute CNS effects, especially the euphoric "flash." This is accomplished by nasal insufflation ("snorting"), intravenous injection, or smoking (free-base form) of the pure compound.

COMMENTS : Amphetamines

The term amphetamine is used variously to indicate a specific chemical agent or to denote a group of compounds that are closely related in both structure and function. The structure of the prototypic compound amphetamine is shown in Figure 1-5a.

² In 1974, these men were in their 20s.

Related agents include methamphetamine ("speed"), ephedrine, methylphenidate, and phenmetrazine. All of these share the basic structural elements of a phenethylamine (see Figure 1-5b), and thus bear great similarity to the neurotransmitters of the adrenergic and dopaminergic systems.



Figure 1-5 a & b: Structures of amphetamine and phenethylamine.

Unlike the endogenous transmitters, which they resemble, the amphetamines are lipophilic, a property that permits facile movement across biological membranes. Absorption from the gut is excellent, and entry into the CNS is unimpeded by the blood-brain barrier. These drugs are effective both orally and intravenously. The latter route provides almost instantaneous access to the brain and more reliably yields the highly prized euphoric "flash."

Metabolic enzymes that process catecholamines (monoamine oxidase) are largely ineffective with amphetamines. Instead, the drugs undergo a variety of catabolic reactions in the liver, including hydroxylation, demethylation, deamination, and conjugation. These operations, however, are relatively slow, and a large part of the drug dose is actually cleared unchanged in the urine. For this reason, and because the amphetamines are bases with high pK_a values, it is possible to accelerate renal excretion by acidification of the urine. In fact, one can remove as much as 80% of ingested amphetamines by this means, a most important consideration in the event of gross intoxication, provided that one remembers that acidification of the urine occurs via acidification of the patient and is not without risk.

Mechanisms underlying the psychomotor effects of amphetamines are referable to catecholamine neurotransmission. To be specific, the drugs enhance adrenergic and dopaminergic activity in the central and peripheral nervous systems by increasing the amount of neurotransmitter released from nerve terminals. This is accomplished with uptake of the drug by the very same mechanisms the catecholamines employ. Once in the terminal, the drug displaces stored transmitter, causing its release into the synapse. It appears that dextro- and levo-rotatory isomers (d- and l-amphetamine) are nearly equivalent in their peripheral actions; however, in the CNS, d-amphetamine is 3-4 times more potent than the l- form. This is due to d-amphetamine's greater ability to activate dopaminergic (e.g., mesolimbic and nigrostriatal) systems, which are so intimately involved with psychomotor function.

Tolerance and Dependence

Tolerance and dependence upon amphetamines have been subject to controversy over the years, yet we know full well that the phenomena do occur with chronic use of the drug. Certain endpoints reflect the development of tolerance most dramatically. For example, the anorexic effect declines rapidly, and is virtually absent at starting doses after a few weeks. Mood altering effects also show a growing dose requirement with time in some users, but not all. With continued administration, an intense psychological dependence manifests itself, especially when the drug is taken intravenously. Physical dependence is characterized by less impressive withdrawal phenomena than those associated with depressants and narcotics, yet is nonetheless distinct. Signs and symptoms include prolonged sleep (which is often disturbed by nightmares--REM rebound), fatigue, lassitude, and depression. Hyperphagia is not uncommon, a point that must be raised when considering the use of these drugs in protracted weight-loss programs. The mechanistic underpinnings of amphetamine tolerance and dependence involve pharmacodynamic compensation within catecholamine systems of the brain. Explicit details of the process await further investigation.

Pharmacological Actions

The systemic actions of amphetamine and its relatives are expressed largely through the CNS, although the cardiovascular system is perturbed as well. Mean blood pressure increases as a result of rising systolic and diastolic pressures. Heart rate often accelerates. However, no change or a decrease may be observed on occasion, due to reflex action.

CNS excitation is apparent in a variety of ways, depending on the dose and route of administration. A relatively low dose of amphetamine (e.g., 10-25 mg) will produce a state of extended wakefulness and decreased sense of fatigue. Both physical and mental abilities are improved, but the effect is strictly a consequence of CNS facilitation, for muscle tone and contractile force are not directly influenced. Mood is usually elevated, often to the point of elation and even euphoria (more likely with intravenous injection than with oral consumption). Interestingly, however, the drug is not a reliable antidepressant. Neurological signs of amphetamine excitation are apparent in EEG tracings, where a wave-form shift towards higher frequency and lower amplitude occurs. With higher doses, frank desynchronization may result.

Amphetamines can produce a certain degree of analgesia, and this property is being exploited in difficult cases of intractable pain relief in order to reduce the opiate requirement. However, chronic use of amphetamines can itself cause pain associated with micturition, for it acts as a sympathomimetic to intensify sphincter tone.

The well-known anorectic attribute of the drug, substantially documented in animal studies, has not lived up to its promise in clinical practice, because it does not provide long-range weight control. We now know that it is inappropriate to employ amphetamines as appetite suppressants for more than a few weeks at a time because of rapidly developing tolerance. Increasing the dose for greater anorectic action may incur a number of grossly undesirable phenomena, including headache and dizziness, palpitation, irritability, suspiciousness, uneasiness, dysphoria, and even delirium.

Chronic abuse of amphetamines frequently leads to psychological disturbance. In fact, the toxic syndrome so closely resembles paranoid schizophrenia that it is difficult to differentiate one from the other. Extreme suspiciousness, paranoid ideation, stereotypic, repetitive behavior, delusions, hallucinations, absence of disorientation, and responsivity to neuroleptics are characteristic of both. The toxic condition can be distinguished from schizophrenia only by the lack of any formal thought disorder, complete recollection of behavior, the appearance of a well-preserved affect, the proclivity for non-auditory hallucinations³ and the presence of amphetamine in the body fluids. Within a matter of days, the toxic condition improves markedly, whereas that of the schizophrenic patient may not [B*Cox, et al., 1983; B*Kalant, 1973; B*Snyder, 1979]. Differences in the presentation may be noted in those who develop the toxic psychosis acutely in contrast to those with gradual onset. The former are less likely to have auditory hallucinations than the latter.

Clinical Usage

When used under the guidance of a physician, prescribed amphetamines may be used in doses of 20-60 mg/day for narcolepsy, 2.5-40 mg/day for hyperkinesis, and 30 mg/day for refractory obesity. Narcolepsy is quite a rare disorder, and can easily be mistaken for other sleep disorders including those caused by other drugs. Hyperkinesis is also rare, but many children seem to be hyperactive and may suffer from attention deficit disorders because of emotional problems stemming from family problems. Refractory obesity means obesity which has not responded to the plethora of safer available treatment modalities such as hypnosis, Weight Watchers, physical conditioning or special diets. Rapid development of tolerance to the anorectic effects of amphetamines limits the usefulness in treating obesity, and the hyperphagia during withdrawal runs the risk of obliterating any gains made while on the drug.

³ While auditory hallucinations do occur in amphetamine psychosis, they are rarely seen exclusive of hallucinations of other sensory modalities, in contrast to paranoid schizophrenia where only the auditory type of hallucinations are usually found.

Amphetamines obtained illegally or from legal sources under false pretenses may be used in ways other than as directed to further performance in athletics, entertainment, scholastics, driving long distances, or working long or irregular hours. If used intermittently in high doses with drug-holidays between episodes, little tolerance may develop, but if used chronically, dosage will need to rise to achieve the desired effect. Seeking sedatives to reverse the stimulation when the need has passed may suggest inappropriate use of stimulants. Doses may exceed 1000 mg/day in amphetamine abusers, so multiple prescriptions and hoarding might be necessary if illicit sources are not used.

Street amphetamines have become rare in the US since the government limited production (see Chapter 7). However, other chemicals are sold as amphetamines such as phencyclidine, phenylpropanolamine, lidocaine, benzocaine, and caffeine. Known as "look-alike" drugs, they are usually packaged in similar capsules and given similar names. Ads for these can be found in porno magazines and other "underground" locations. Physicians are still prime targets for the amphetamine abuser, although many physicians are much more cautious about prescribing them than in the past. Narcotics agents monitor prescribing practices and periodically indict a physician for improper prescribing. The patient who presents to the physician requesting amphetamines probably will be quickly recognized, however some are fairly crafty, claiming to have been on amphetamines from their local doctor, but having lost them or run out of them while traveling. Sometimes patients will obtain prescriptions for others or for sale on the street, in which case their urine toxicology screen will be negative despite claiming to have used up the last prescription. Some patients will be filling prescriptions from several doctors, sometimes at only one or two pharmacies. Asking which pharmacy they use, at each visit, and then calling the pharmacist(s) to learn of the patient's total drug profile is sometimes quite revealing. The urine toxicology screen may also reveal other drugs not mentioned in the interview, some of which might even explain the symptoms being treated. Patients who obtain small prescriptions for amphetamines, but who hoard them for recreational sprees, will also have negative toxicology screens despite claiming to be taking them regularly. Verification of history by family or significant others may also reveal dishonesty or concern about the patient's drug abuse problem. Careful documentation of the size and frequency of prescriptions, calling the pharmacy to verify that the prescription was not altered, keeping close control of prescription blanks, and refusing to issue refills over the phone (even when the pills allegedly fell in the toilet) may be useful techniques for avoiding becoming an enabler for a drug dependent patient [J*Bentorado, 1980; J*Scheibel, 1980]. In short, the doctor may have to play detective if the patient is lying about patterns of usage or motivation for use.

TEACHING HINTS: Amphetamines

Have the students read about and then discuss the diagnostic distinction between amphetamine psychosis and paranoid schizophrenia. Have them discuss the differing presentations of an amphetamine abusers who obtain them from physicians, family, friends, or street pushers [B*Brecher, 1972; B*Cohen, 1981; B*Cox et al., 1983; B*Hofmann & Hofmann, 1973; B*Kalant, 1973; B*Mandell, 1976; B*Ray, 1978; B*Shick, Smith & Wesson, 1973; B*Spotts & Spotts, 1980].

Have the students find some magazine ads for "look-alike" drugs and have them discuss ways they might distinguish these from true amphetamines if an adolescent gave a history of taking "black beauties" obtained illicitly.

Have the students discuss the approach they would take to patients presenting with hypersomnia, obesity or childhood hyperactivity. How would they diagnostically evaluate these cases? What clinical treatments would they try before beginning amphetamines? Show videotapes of drug abusers discussing how they try to fool doctors into giving them drugs [A*Doctor, you've been lied to; A*Feelin' no pain; A*Gallant, Alcohol and Drug Abuse among Physicians; A*Substance abuse and the physician; A*Some of the things that go on out there]. Or have them read Nightmare Season [B*Mandell, 1976] and then discuss the use of stimulants by professional athletes.

EVALUATION: Amphetamines

In clinical precepting of residents, how do they approach the evaluation of patients who request or hint at the desire for stimulants? Or those who present with hypersomnia, obesity or childhood hyperactivity? Does the resident routinely obtain a urine specimen for toxicology during this evaluation? How long does the resident delay prescribing amphetamines? How does the resident monitor use of them to be certain that they are being used appropriately?

COMMENTS: Cocaine

Obtained from the Andean shrub *Erythroxylum coca*, cocaine is imbued with local anesthetic as well as sympathomimetic properties. The molecular structure of this drug more closely resembles that of lidocaine than the catechols (see Figure 1-6). In contrast to amphetamines, only the dextrorotatory configuration of cocaine is viable as a psychomotor stimulant. Material procured on the street, it should be noted, may contain as little as 10% or as much as 90% of the pure drug.

Illicit cocaine is commonly adulterated with local anesthetics (e.g., lidocaine or procaine) and amphetamines that look and act like cocaine. Sugars, such as lactose, may also be added as inert filler. Absorption is not impeded by membrane barriers,

and thus a variety of administration modes are employed. Preeminent among these are oral consumption, intravenous injection, insufflation ("snorting"), and smoke inhalation. The rate of absorption follows this order closely, with onset of drug effects occurring several minutes after administration by mouth, but after only a few seconds after smoking the free-base (uncharged) form [J*Smith, 1984]. Absorption of the orally ingested drug is greatest in the mouth. It is trapped in the stomach as a charged, basic molecule by the acidic environment.

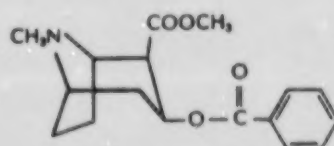


Figure 1-6: Structure of cocaine.

Chemistry and Metabolism

Demethylation is the primary metabolic reaction that cocaine undergoes in the liver. One immediate product, norcocaine, is as active as the original compound, but others, such as benzoylecgonine and ecgonine, are inactive. The latter are highly polar compounds and so are rapidly cleared in the urine. Cocaine's duration of behavioral action is approximately 30 minutes.

The precise mechanism by which cocaine elicits psychomotor activation and the euphoric "high" has long been ascribed to cocaine's sympathomimetic quality. However, recent clinical research suggests that the local anesthetic action may be of greater importance. In blind studies with cocaine-familiar subjects, the researchers found that lidocaine applied nasally was as efficacious a euphorigenic as cocaine, and therefore, indistinguishable from it. The neuropharmacologic rationale for this at a cellular level is unknown. Mechanistically, one can make a far better case for cocaine action via sympathomimetic processes, for it is well known that the drug potentiates endogenous catecholamine function by inhibiting transmitter reuptake into presynaptic terminals and prolongs the synaptic action of these neurohumoral agents. Speaking against this thesis, however, is the fact that drugs with similar sympathomimetic action, for example, tricyclic antidepressants, are not euphorigenic. Cocaine, like amphetamines, is not a reliable antidepressant, although it does improve mood state for many people.

Tolerance and Dependence

Tolerance and dependence are engendered by chronic use of cocaine. These phenomena are most apparent in cases of consistent, high dose consumption, especially free-base smoking, and generally resemble those described for amphetamines.

Pharmacological Effects

The acute and chronic pharmacologic effects of cocaine are not unlike those of amphetamines, with the exception of its additional local anesthetic action. Thus, systemic cardiovascular performance may be modified by enhanced peripheral vascular tone and accelerated, desynchronized cardiac rhythm. Indeed, toxic amounts of cocaine may induce fatal arrhythmias. The CNS is likewise subject to the local anesthetic-imposed toxicity, including frank convulsions of the tonic-clonic type.

TEACHING HINTS: Cocaine

Have the students read about cocaine [B*Brecher, 1972; B*Byck, 1974; B*Eiswirth, Smith & Wesson, 1973; B*Hofmann & Hofmann, 1975; B*Mandell, 1976; Petersen & Stillman, 1978; B*Ray, 1978; B*Schoener, 1981; B*Schuckit, 1979; J*Smith, 1984; B*Wilford, 1981] or watch a film [A*Cocaine] before discussing its use and abuse in this country.

HALLUCINOGENS

COMMENTS: Hallucinogens

This class of drugs is rather heterogeneous when considered in a mechanistic sense, yet all agents in the group share a common capacity to elicit vivid hallucinations. Various descriptive terms have been applied to the class, including "psychotomimetic," "psychogenic," "psychedelic," "deliriant," but none of these is truly accurate or appropriate, and so "hallucinogen" remains the accepted term.

Primitive peoples have utilized "mind- or consciousness-expanding" agents for millennia in healing, mystical, and religious rites and, in this context, the use has not proven deleterious. We have had quite a different experience in the United States, where hallucinogen abuse has been taken to the extreme. The postwar years of the 1950s ushered in the "psychedelic generation" of adventurous students, artists, and intellectuals who pursued nirvana through these exotic drugs. Although the fervor of that age has now subsided, the attraction of hallucinogens persists for many. In fact, the demographics of hallucinogen use have changed over the past 30 years to encompass a far broader representation of the American population. However, chronic abuse of these substances is most uncommon and the frequency of use seldom exceeds 3-4 times per month. Marijuana use is exceptional in this regard, but the usual dose and frequency of consumption is not typically associated with an hallucinogenic experience [B*Brecher, 1972; B*Hoffman & Hofmann, 1975; B*Ray, 1978].

Factors of set and setting play a major role in the individual's response to hallucinogens. Certainly, the unique effects depend greatly upon the particular drug employed and the amount taken, but the user's preexisting state of anticipation and knowledge, as well as the warmth and familiarity of the surroundings, color and shape the experience. Members of the psychedelic drug subculture are well aware of this, and seldom induct a new comrade without the aid of a "guide" who has been there before.

Hallucinogen chemistry is diverse, including LSD, PCP, marijuana, and other drugs commonly on the scene today. Some of these agents are indole or tryptamine derivatives, whereas others are more closely akin to phenylethylamines (e.g., amphetamine). Still others are frankly known to act like or antagonize acetylcholine or GABA neurotransmission in the brain. Because no common denominator exists, it would best serve the purposes at hand to summarize briefly some pertinent details about each of the substances encountered most frequently.

LEARNING OBJECTIVES: LSD

The learner should be able to (1) discuss the chemistry of LSD; (2) describe the absorption, distribution, and elimination of LSD; (3) describe the autonomic and somatic motor responses to LSD; and (4) describe the hallucinogenic experience as characterized by LSD use.

COMMENTS: LSD

If any one agent in the group might be prototypic, it would be Lysergic Acid Diethylamide (LSD). This material is found in nature as an ergot derivative manufactured by the fungus that grows on rye and other grains. It is closely related to drugs such as ergonovine, which is employed therapeutically for its contractile effect on smooth muscle. The natural source, however, is not the usual one. LSD is normally synthesized in the laboratory, and was first introduced by Albert Hofmann, a Sandoz chemist, in 1943.

Chemistry & Pharmacology

The structure is complex, as illustrated in Figure 1-7, but it contains a recognizable indole nucleus, which may be responsible for specific binding to neurotransmitter receptors. Over the years, a plethora of LSD analogs has been synthesized for the legitimate purpose of pharmacodynamic study. At one time it was believed that LSD produced "a model psychosis" and so it was theorized that a careful investigation of its chemical structure and mechanism of action would reveal the etiology of the psychotic disorders.

Unfortunately, this hypothesis has not been borne out, but much has been learned about the structure-activity relationship of LSD. For example, potency of the compound is enhanced by addition of nitrogen substituents. The most potent form, LSD-25, has a pair of ethyl groups on the amide nitrogen. The CNS effects are produced only by the dextrorotatory stereoisomer, although the levorotatory one exerts considerable systemic action.

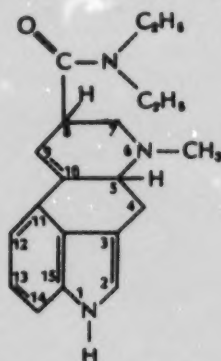


Figure 1-7: Structure of LSD.

LSD is extraordinarily lipophilic. Thus, when taken orally in the usual manner, it crosses membrane barriers with facility, and rapidly accumulates in the brain. Extremely low doses are required for CNS effect; as little as 50-100 microgram is sufficient to elicit a fully developed hallucinogenic episode. LSD's plasma half-life has been quantitatively determined to be approximately 3 hours. Hepatic oxidation of the compound is primarily responsible for its demise. The process is virtually complete, and inactive products are excreted in both urine and bile.

Although studied repeatedly, the molecular pharmacodynamics of LSD remain incompletely defined. At a cellular level, the drug is known to have great affinity for serotonergic receptors and to act upon serotonin-containing cells of the brainstem. Axodendritic synaptic function is apparently depressed by LSD, whereas axosomatic transmission is enhanced. The significance of this is unclear at present.

Clinical Effects

A wide range of neurologic and psychologic effects are evoked by LSD [B*Fisher, 1968; B*Ungerleider, 1968]. The autonomic nervous system exhibits primary central activation of both parasympathetic and sympathetic outflows. The sympathetic facilitation predominates, with obvious pupillary dilation, hyperthermia, tachycardia, piloerection, and hyperglycemia. However, at excessively high doses, effects on the medullary brainstem may result in cardiovascular and respiratory depression. Major somatic motor systems are affected as well, with the appearance of tremor, hyperreflexia, ataxia, and spastic paresis. Concurrent psychological manifestations include excitation, labile mood, cognitive dysfunction, perceptual distortion, hallucination, depersonalization, and schizophreniform behavior.

The typical LSD "trip" lasts about 8-12 hours, and occurs in roughly three overlapping stages: (1) somatic symptoms (dizziness, drowsiness, paresthesia, and tremors) are followed by (2) perceptual distortion that is largely visual and is ultimately superceded by (3) total psychic modulation (disturbance of time/space perception, depersonalization). Within just a few minutes of oral ingestion of the drug, a sense of total relaxation envelops the person, and the mood becomes labile. Autonomic symptoms next make their appearance and often prove disconcerting, especially to the novice. Panic reactions at this point are not uncommon. Within a half-hour's time, perceptual change ensues. Visual perception is markedly altered, with bright, colorful images passing through the field. The user often concentrates inordinately upon microscopic detail. Images persist and merge as the focus of attention slowly shifts. Other sensory modalities become involved, and synesthesia (or the melding of different modalities) can occur. Sounds may be visualized and colors smelled. Size, direction, and distance are distorted, and time seems to halt its progress as the user slips into a fantasy world. Emotions are deeply affected, but vary with the person. True ecstasy or sheer horror are equally possible. In spite of this emotional upheaval, the user remains outwardly impassive, often sitting or lying down with the eyes closed. As the trip approaches its quintessence, ego boundaries dissolve and aspects of self merge into the external world. Upon conclusion of the experience, the exhausted "tripper" nods off and may sleep for a day [B*Ungerleider, 1968].

Tolerance to the pharmacologic effects of LSD is well documented. However, behavioral as well as pharmacodynamic processes contribute to the phenomenon. The response to repeated doses is rather complex, due to the variable facets of set and setting. Strict dose-effect relationships are difficult to compute. Interestingly, cross-tolerance between LSD and the chemically dissimilar substances psilocybin and mescaline is evident. The habitual LSD user would, therefore, require a larger-than-usual dose of these drugs to provide a satisfying "trip."

Toxicity

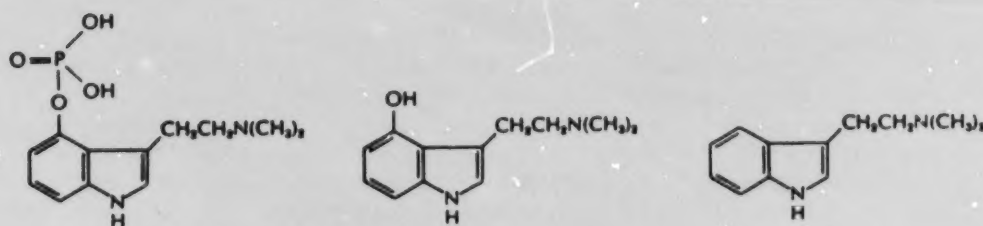
With regard to toxicity, it is noteworthy that the "therapeutic ratio" for LSD is high, that is, the LD₅₀ is much larger than the ED₅₀. Lethality as a direct consequence of drug action has never been reported. Fatal accidents certainly may occur through secondary drug effects on perception and judgement, due to the greatly compromised physical and mental ability. Occasionally, overdoses have resulted in persistent psychotic behavior, hyperkinesia, catatonia, and coma. "Flashbacks" or "free trips" have also been reported by those who have taken LSD and other related hallucinogens several times. These experiences are essentially drug-free replicas of the drug-induced trip. However, they are usually briefer and often disconcerting because the person is unprepared (set and setting inappropriate) [B*Fisher, 1968].

LEARNING OBJECTIVES: Psilocybin family

The learner should be able to (1) describe the source and chemistry of psilocybin and related compounds; (2) describe the absorption, distribution, and elimination of psilocybin; (3) list the autonomic and psychological effects of psilocybin; and (4) compare the psilocybin "trip" with that of LSD.

COMMENTS: Psilocybin family

Psilocybin and psilocin, a closely related alkaloid, occur naturally as constituents of the mushroom *Psilocybe mexicana* and, to a lesser extent, in other mushroom species (see Figures 1-8a & b). In fact, once ingested, the psilocybin is rapidly hydrolyzed to psilocin, the 4-hydroxylated ester of dimethyltryptamine (DMT; see Figure 1-8c). Upon recognition of the fact that the substituted methyltryptamine was itself an active compound, chemists of the psychedelic subculture immediately proceeded to synthesize a number of such agents, including alphamethyltryptamine (AMT), demethyltryptamine (DMT), and diethyltryptamine (DET). All of these presumably have similar modes of action involving serotonergic transmission in the brain.



Figures 1-8a: Psilocybin; 1-8b: Psilocin; 1-8c: N,N-dimethyltryptamine (DMT)

Clinical Effects

Behavioral and autonomic manifestations of this group are remarkably similar to those of LSD. Sympathomimetic sequelae include mydriasis, hypertension, and tachycardia. Psychological effects consist of anxiety, perceptual distortion, and hallucination. Differences do exist, however, in that certain members (e.g., AMT) have more pronounced somatic actions, whereas others (e.g., psilocybin) favor the central effects. All are absorbed through the gut. They achieve effective plasma concentration by this route within 30 minutes and so are generally taken by mouth. However, they can and have been administered parenterally for

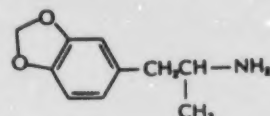
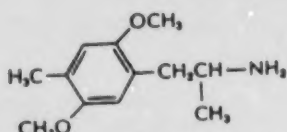
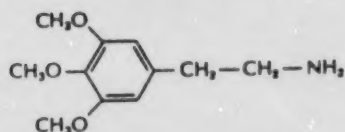
more rapid onset and greater potency of action. Duration of action is variable for the different compounds by any given route of administration. For example, orally consumed psilocybin will induce a syndrome lasting approximately 3-4 hours, whereas the syndrome of AMT may persist for more than 12 hours. Hepatic metabolism accounts for primary termination of action by oxidative deamination and hydroxylation. The catabolic products are more polar than the parent compounds and thus are more rapidly excreted into the urine. Because they are bases with relatively high pK_a values, clearance may be accelerated by acidification in cases of gross intoxication.

A decrease in the hallucinogenic effects of psilocybin with repeated administration has been demonstrated in clinical investigation, but it seems to develop more slowly than does that for LSD. Tolerance to the actions of AMT, DMT, and DET has not yet been established. As noted above, psilocybin and LSD may engender a significant degree of cross-tolerance. Psychological dependence, characterized by an intense craving, is possible with chronic psilocybin use, whereas physical dependence may not occur.

The toxicity of these tryptamines has not been systematically investigated. However, anecdotal reports suggest that extremely large doses may prove fatal by intensified autonomic action, for example, hyperpyrexia, leading to convulsions and cardiac arrhythmogenesis.

LEARNING OBJECTIVES: Mescaline family

The learner should be able to (1) describe the source and chemistry of mescaline and related compounds; (2) describe the absorption, distribution, and elimination of mescaline; and (3) compare autonomic and CNS actions of mescaline with those of LSD.



Figures 1-9a: Mescaline; 9b: DOM (STP); 9c: MDA

COMMENTS: Mescaline family

The peyote cactus, indigenous to northern Mexico, synthesizes a hallucinogenic substance called mescaline, which has been part of Native American life for centuries. As in times past, the natural material is used to enhance tribal rites and

religious celebration, to relieve hunger and fatigue, and to treat bodily ailments. In the United States, this use is currently protected by law and is limited to members of the Native American Church.

Chemistry & Pharmacology

Mescaline is unlike the preceding hallucinogens in its chemical makeup (see Figure 1-9a). Indeed, as a phenylethylamine derivative, it more closely approximates the structure of amphetamine and the catecholamines than it does LSD or the tryptamines. Since the identification of its chemical composition (trimethoxyphenylethylamine), creative chemists have synthesized a number of analogs similarly imbued with hallucinogenic potential. These include dimethoxymethylamphetamine (DOM or STP; see Figure 1-9b) and methylenedioxymphetamine (MDA; see Figure 1-9c), drugs that experienced their heyday in San Francisco during the 1960s, but sustain their interest today among a small, dedicated group of street pharmacologists.

Most commonly, these substances are taken by mouth, but parenteral administration is not unknown. Absorption from the gut is excellent, and peak blood levels of mescaline are attained within 2 hours after ingestion. Brain concentration of the agent follows blood level closely. With a plasma half-life of 6 hours, mescaline exerts its neuropsychologic effects for as long as 12 hours. A majority of the material is eliminated in active form by renal excretion, whereas the remainder is subject to oxidative deamination in the liver. Disposition of DOM is like that of mescaline, though more rapid and so its effects often resolve within about 6 hours.

The psychedelic syndrome produced by mescaline and the amphetamine analogs is clinically similar to that of LSD or the tryptamines, yet the chemical constitution of these compounds suggests a very different mode of action. Whereas serotonergic mechanisms are implicated in LSD actions, one would surmise that catecholaminergic processes are inherently more important for these drugs. This has yet to be verified in the laboratory.

Clinical Effects

Stimulation of autonomic and somatic motor systems is an early manifestation of mescaline intoxication. Sympathetic activation (mydriasis, hypertension, and tachycardia), hyperreflexia, and static tremor reflect this facilitation. Nausea and vomiting occur at this time, more frequently than with LSD. Vivid visual illusions are most characteristic of the mescaline experience, though true hallucinations may never occur. Anxiety and depression, hallmarks of amphetamine action, occur with some regularity when mescaline is chronically consumed. Tolerance develops under these conditions and, remarkably, so does cross-tolerance with LSD and psilocybin. Physical dependence has not been detected, but psychological craving is definitely associated with repetitive use of these phenylethylamines.

LEARNING OBJECTIVES: Phencyclidine

The learner should be able to (1) describe the chemistry and patterns of use of PCP and its analogs; (2) discuss functional aspects of PCP; (3) list at least 8 manifestations of "low-dose" intoxication with PCP (5 mg); and (4) describe effects of higher-dose intoxication with PCP.

COMMENTS: Phencyclidine

Parke, Davis and Co. developed phencyclidine (PCP) in the late 1950s as a novel anesthetic agent that would provide surgical anesthesia without generalized depression. Brief clinical trials immediately revealed drawbacks to this therapeutic application (persistent hallucinations after surgery) and the drug was quickly withdrawn from the market. A congener of PCP, ketamine, was independently marketed by the same pharmaceutical company for pediatric anesthesia, and though it has been used for a number of years with some success, indications for it remain limited.

With the first report of its hallucinogenic properties, adherents of the drug subculture seized upon PCP as a major addition to their pharmacopoeia. They quickly learned its rather fundamental chemical synthesis and made it widely available in liquid, powder, and tablet form. As a result of this unfettered distribution and use, we have learned a great deal about the pharmacologic and toxic actions of the drug in the intervening years. Unfortunately, PCP is often misrepresented when sold on the streets or added to preparations of other substances, and the user may be totally unaware that "angel dust" is being taken [B*Linder, Lerner & Burns, 1981].

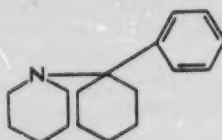


Figure 1-10: Phencyclidine

Chemistry & Pharmacology

PCP is one of a group of cyclohexylamine compounds that share a common neurophysiologic potential. More than 30 analogs are now known and sold under the "trade names" of PCC, PCE, PHF, TCP, etc. Most of these contain a benzene ring, a phenyl group, and a secondary or tertiary nitrogen (often in a ring structure, e.g., piperidine; see Figure 1-10). Because the quality control maintained by illicit labs is not equal to that of our legitimate pharmaceutical industry, impurities are often found in PCP preparations, including potassium cyanide, which is employed in

the synthetic process.

In spite of the high pK_a of this base (9.43), gastrointestinal absorption is adequate to provide the sought-after effects, and so the drug is most frequently taken by mouth. However, it was recently observed that inhalation of the material by smoking yields more rapid onset of action and greater ability to titrate the endpoint. Hence, this route of administration has achieved growing popularity. Biotransformation of PCP is accomplished in the liver, primarily by hydroxylation to mono- and dihydroxy products, which are then conjugated with glucuronide and other compounds to allow more facile urinary excretion.

Clinical Effects

Pharmacologically, PCP is multipotential. It has stimulant, depressant, and hallucinogenic properties, depending on the amount of the drug taken. This mosaic of activity suggests that the drug interacts with several neurotransmitter systems. Evidence to support such a thesis has been generated in the experimental laboratory. Dopamine, acetylcholine, serotonin, and other transmitter systems are disturbed by PCP, but the most specific actions seem to be associated with cholinergic (muscarinic) and opioid substrates. PCP actually binds to receptors for those transmitters. In addition, it now appears highly likely that a unique receptor for PCP exists in brain tissue. This is a fact that raises most intriguing questions about an endogenous ligand for such a site, its role in (ab)normal brain function, and the possibility for developing a specific drug antagonist.

Autonomic and somatic motor systems are affected in a cocaine-like fashion at a relatively low dose, resulting in hypertension, tachycardia, agitation, and other sympathomimetic or psychomotor manifestations. Further intoxication yields a "drunken" anesthetic state in which the extremities become numb, perception is distorted, and the user experiences sensory isolation. [J*Aronow & Donel have identified characteristic signs and symptoms of PCP intoxication according to the degree of intoxication. At blood levels of 20-30 ng/ml (dose of approximately 5 mg), they find the user shows some of the following symptoms: agitation; motor incoordination; impassive appearance; catatonic rigidity; catalepsy; nystagmus, vertical or horizontal; loss of pin-prick response; flushing; diaphoresis; or hyperacusis.

Accompanying these signs and symptoms are cognitive disorganization, feelings of estrangement and apathy, and changes in body image. Bizarre and hostile behavior is entirely likely. Doses that lead to blood levels of 30-100 ng/ml may result in the eyes being open; nystagmus; hypersalivation; vomiting; stupor or coma; muscle rigidity; diaphoresis; pyrexia; stereotypy; or decrease of all sensation. Even greater intoxication (blood levels of greater than 100 ng/ml) will potentially induce sustained coma; hypertension; opisthotonic spasm; decerebrate rigidity; muscle spasm and involuntary movement; loss of sensation; loss of corneal and gag reflexes; diaphoresis; flushing; or

hyperpyrexia convulsions. Blood levels above 200 ng/ml is associated with respiratory depression and above 500 ng/ml with death [B*Linder, Lerner & Burns, 1981].

A life-threatening condition may persist for days or weeks after PCP poisoning. Successful treatment requires vigilance, supportive measures, reduction of the drug load by gastric lavage, and ion trapping with vigorous acidification of the urine. Permanent physical and mental disabilities after PCP poisoning have been reported. Spinal fluid levels may remain elevated for weeks after ingestion, though serum levels may be undetectable. However, qualitative identification may be achieved by measuring gastric fluids (very acidic) or urine following an acid load. When a sample is taken, one should measure and record the pH immediately, since the quantitative serum:fluid ratio depends upon pH.

Clinical Management

Neuroleptic treatment for the delirium is contraindicated. Chlorpromazine potentiates the convulsant action of PCP and competes in the kidney with phenacyclidine for excretion. A dark quiet room, and if necessary, sedation with diazepam may be beneficial during the acute intoxication, provided the patient is maintaining respiratory function adequately. "Talking the patient down" does not help, in contrast to LSD and mescaline family drugs.

TEACHING HINTS: Phenacyclidine

Have the students watch a film [A*Angel Death] before discussing the acute management of an acutely intoxicated agitated patient.

MARIJUANA

LEARNING OBJECTIVES: Marijuana

The learner should be able to (1) describe the patterns of marijuana use, including its different forms; (2) describe the source and chemistry of marijuana; (3) discuss absorption, distribution, and elimination of marijuana; (4) describe the acute CNS effects of marijuana; (5) list at least 6 toxic effects of marijuana overdose; (6) discuss the relationship of marijuana to psychiatric disorders; (7) describe effects of delta-9-THC on the cardiovascular and respiratory systems; and (8) discuss possible therapeutic actions of delta-9-THC.

COMMENTS: Marijuana

Marijuana is truly in a class of its own. It is presented here because of its ability to induce an hallucinogenic syndrome at high dosage, although it is seldom used in this manner.

Marijuana is one of the oldest drugs known to humanity. An extract from the flowering tops of the annual plant *Cannabis sativa*, in years past, it was used for a great variety of medical complaints. Its current role is more narrowly defined, no longer sanctioned by society. Yet, in spite of this illicit status, more than 50 million Americans report that they have at least tried the substance. Approximately two-thirds of the 18-25 year-old population has used marijuana, and this young adult group is most likely to indulge on a regular basis. Surveys of high-school seniors indicate that approximately one in ten students smokes "pot" daily [B*Abel, 1980; B*Bloomquist, 1971; B*Brecher, 1972; B*Erickson, 1980; B*Fehr & Kalant, 1984; B*Goldman, 1979; B*Hendin et al., 1981; B*Hofmann & Hofmann, 1975; B*Kalant et al., 1984; B*Ray, 1978].

Chemistry & Pharmacology

The primary active principle in marijuana is the cannabinoid delta-9-tetrahydrocannabinol (delta-9-THC). However, the plant material contains 61 known cannabinoids, and hundreds of other chemical substances, some of which are bioactive. In order to produce the characteristic CNS actions of marijuana, the substance must contain (1) a three-ring structure, including a pyran ring; (2) a free phenolic hydroxyl; and (3) a lipophilic side chain (see Figure 1-11). The concentration of delta-9-THC is variable, depending on the source of the material, and usually ranges from 0.5-2.0% of the total leaf mass. Hashish, a more concentrated form of the leafy resin, contains between 5 and 10% delta-9-THC; further refined hashish oil has 30% or more of the active agent.

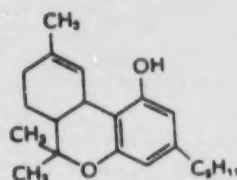


Figure 1-11: Delta-9-THC

When marijuana is smoked, as is usually the case, the composition is subject to pyrolysis. However, some of the delta-9-THC loss is offset by thermal activation of other cannabinoids. Approximately one-half of the active drug is absorbed in the process of smoking. The balance is lost by entrapment in the butt ("roach"), in smoke not inhaled, in smoke exhaled, and through pyrolysis.

The oral route of administration, which preserves nascent delta-9-THC, is actually less efficacious than inhalation of the combustion product, for a number of reasons. Primary among these are the very extensive and well-perfused surfaces available for absorption in the lungs, and the hepatic catabolism of the drug taken up in the mesenteric circulation ("first pass effect").

Delta-9-THC is highly lipid soluble. Therefore, tissue distribution and cellular accumulation are rapid. Polyphasic elimination kinetics indicate that the drug undergoes redistribution after the initial uptake and is stored in lipid depots for long periods of time. The remnants of a single dose can be detected in blood samples for up to one month. Plasma half-life values for delta-9-THC vary from 19 to 57 hours, but may actually be longer. Catabolism, primarily by hepatic hydroxylation, is rapid and essentially complete, the bulk of catabolites exiting fecally.

Although a wealth of empirical information about the body's response to marijuana is readily at hand [B*Fehr & Kalant, 1984; B*Kalant et al., 1984], we are woefully deficient in our understanding of the fundamental mechanisms responsible for these effects. We do know that cannabis inhibits amino acid incorporation into protein and thereby alters protein synthesis, but this is more likely responsible for chronic toxicity than acute pharmacodynamics. Since neurotransmission is the currency of brain function, we should address our mechanistic inquiry to the interaction between marijuana and the chemical neurotransmitters. Studies on catecholamines, serotonin, and GABA, however, have revealed little encouraging data. Only acetylcholine (ACh) has proven sufficiently responsive to the drug for serious consideration. Apparently, delta-9-THC causes a reduction in acetylcholine turnover (synthesis, release, and degradation), reflecting diminished cholinergic neuronal activity. Acetylcholine, it should be recalled, is one of the brain's major excitatory neurotransmitters.

Clinical Effects

Most people smoke marijuana for the sense of well-being and euphoria they obtain, but they may experience some dizziness, paresthesia, and even dysphoria on occasion. Normal levels of consumption lead to a dramatic slowing of time perception, although visual and auditory senses remain intact, while the user enters a dream-like state. Cognitive function is disturbed; mentation and intellectual expression both come with difficulty, and anterograde amnesia may be evident. Feelings of isolation and depersonalization are not infrequent. Large doses produce a variety of disconcerting symptoms, including visual and auditory perceptual distortion. Even larger, toxic amounts of the drug may cause incapacitating motor and visual effects: incoordination, ataxia, tremors, dyskinesia, mydriasis, nystagmus, photophobia, and headaches. These symptoms are pathognomonic for cannabinoid poisoning when adequately differentiated from potential traumatic or other drug-related insult.

Chronic, regular use of marijuana in moderate amounts alters waking and sleeping EEG patterns; stage IV sleep is increased, though REM is reduced. However, there is little evidence to support the contention that even heavy, chronic use of the substance will cause cerebral atrophy, as once was claimed.

Although difficult to substantiate, psychiatric illness has frequently been attributed to marijuana use. Acute panic states are not uncommon, especially for naive or unsuspecting users, but this toxic condition wanes with drug elimination. The exacerbation of preexisting, perhaps latent, schizophrenic syndromes may account for some reported psychiatric consequences. A true toxic delirium is not unlikely. Frequently mentioned in the clinical literature is a depressive condition called the "amotivational syndrome." It is characterized by complacency and disinterest, isolation and a loss of drive to succeed. Well-controlled studies do not lend credence to its existence as a unique condition, but the practitioner should remain aware of its potential.

Many bodily functions other than those of the CNS are influenced by marijuana. Prominent among these are cardiovascular, respiratory, visual, and hormonal functions. Some effects are clearly detrimental, whereas others may be beneficial. The most obvious manifestation of marijuana on cardiovascular performance is a dose-related, sinus tachycardia due to increased sinus automaticity, sinoatrial and atrioventricular nodal conduction. Propranolol (Inderal [R]) will effectively counter this response, but the changes seldom compromise cardiac performance. Large doses may have a direct impact on the vasculature, causing hypotension. Respiration may well suffer the adverse consequences of smoking per se, for the constituents of marijuana smoke are extremely irritating to the bronchopulmonary epithelium. Regular, heavy marijuana users often complain of pharyngitis, rhinitis, bronchitis, and even asthma [J*Tennant, 1980]. Delta-9-THC has a compensatory smooth muscle relaxing property that promotes bronchodilation. However, it is subject to tolerance and so accedes to the adverse epithelial effects with repetitive use.

Several visual consequences of marijuana use have been recounted, and two points should be raised in this regard. One is the dramatic engorgement of conjunctival vessels, producing a characteristic bloodshot appearance (not subject to tolerance), and the second is a reliable decrease in intraocular pressure. The latter effect of delta-9-THC is shared by other cannabinoids, and is being explored for its therapeutic value in the management of glaucoma.

A sexual mystique surrounds the use of many abused drugs, and marijuana is no exception. However, clinical experience has shown that it may be as problematic in this context as often as it is satisfying. Low doses of the drug seem to be disinhibiting, and thereby encourage sexual activity in much the same fashion as alcohol. Higher doses diminish sexual interest and performance. Sex steroids (testosterone, FSH, and LH) are significantly depressed. Gynecomastia is seen in males who use the drug with regularity. A number of other seemingly unrelated marijuana ef-

facts are noteworthy for their toxic or therapeutic potential. For example, studies have shown that the drug may be both mutagenic and carcinogenic under certain conditions. Until we learn more about these effects, one should certainly caution patients about the prospective consequences. Food consumption is increased significantly. Anecdotal reports to this effect (bouts or the "munchies") have been substantiated in controlled experiments. With this in mind, cancer patients have been tested and respond well to treatment with delta-9-THC, appetite is increased, and weight is gained. Furthermore, the chemoreceptor trigger zone of the brain is suppressed by marijuana, thus the emesis that results from radiotherapy activation is suppressed. Analgesia comparable to that of the opiates can be obtained with delta-9-THC, but tolerance to this develops with frequent use. An opioid mechanism may be implicated here, and so conjoint use of marijuana and opiates would not yield potentiation.

People who smoke marijuana regularly tend to increase their intake with time. This is an expression of tolerance to the euphorogenic action. The tachycardia also shows tolerance, as do many of the other effects. Both dispositional (increased metabolic rate) and pharmacodynamic mechanisms contribute to this phenomenon, although the latter seems to be of greater significance.

Tolerance & Dependence

Dependence on marijuana has long been a subject of controversy. That psychological dependence occurs is no longer debatable, but the presence of physical dependence is still in dispute. Clinical researchers supporting the notion point out that users experience definite hyperirritability and even dysphoria between doses. Opponents suggest, however, that these minor signs are not sufficiently discrete to establish an abstinence syndrome.

INHALANT ABUSE

LEARNING OBJECTIVES: Inhalants

The learner should know the major types of abusable inhalants and their sources, side effects, dangers, and the population at highest risk of abuse.

COMMENTS: Inhalants

The inhalants considered in this chapter are a diversified group of chemicals. They vaporize to a gaseous state at room temperature, and their vapors, when inhaled, produce mind-altering effects. Most people do not even consider these substances to be drugs, because they are not usually manufactured for this pur-

pose. Substances inhaled for their psychoactive effects include solvents, aerosol sprays, anesthetics, and volatile nitrite "room odorizers." Inhalation of psychoactive drugs via "snorting" or smoking has been known since antiquity. The ease of administration, rapid onset of action, and control over the dose are all factors that encourage this route of administration. Only recently has technology added volatile substances and gases to the stock of mind-altering drugs that can be taken via inhalation.

The inhalants used to get "high" are commercially available agents normally used for cleaning, or as beauty agents, fuels for motor vehicles, glues, or paint thinners. Few volatile agents (only the most toxic) have escaped being used by those searching for mind-altering inhalants. Most of these agents are considered to be "safe" by users and, indeed, many of the agents have been used as directed for decades without being implicated as serious health hazards. The agencies charged with mandating appropriate use of these products have focused their attention on preventing acute intoxication during normal use. Consequently, little attention has been paid to the problem of intentional inhalation of these substances for recreational or mind-altering purposes. Required labels warn normal users about acute exposure and intoxication from inhaling the fumes, whereas the problems associated with chronic "sniffing" continue to be downplayed or ignored by manufacturers who feel that their liability is limited to those risks encountered when their products are used according to the label instructions.

Many of the agents used preferentially by inhalant users are readily available because of the widespread use of such agents in the home and at work. Thus, inhalants provide a cheap, available, and usually legal means of getting "high." Because of their ready availability, they have been used experimentally by young people. Use often begins in the preteen years, particularly among people of lower socioeconomic status who cannot afford other recreational drugs, such as marijuana or alcohol [B* Cohen, 1977].

PATTERNS OF USE

Recreational use of inhalants began in the early 1800s. The first agents used in this manner were the anesthetics: nitrous oxide, chloroform, and ether. Recreational use began soon after their discovery, and was followed by their use in the fields of surgery and dentistry once their analgesic effects had been noted. The recreational use of anesthetics continued in Europe and America throughout the remainder of the nineteenth century, and there have been occasional resurgences during this century. At present, nitrous oxide is a popular agent with certain segments of the population.

Among the earliest solvents used were various petroleum distillates, especially gasoline. Glue-sniffing appeared as a widespread phenomenon in the late 1950s; since the early 1960s, the practice of using inhalants has spread across a broad spectrum of products that contain volatile organic compounds.

It has been estimated that approximately 7 million people in the United States have used or experimented with inhalants. Although use varies with the geographic regions studied, in most surveys, slightly less than 1% of surveyed adolescents reported current or continued use of inhalants. In these same surveys, persons responding indicated that approximately 7-9% of 12-17-year-olds and 11% of 18-25-year-olds had tried inhalants at one time or another [B*Carroll, 1977]. These data indicate that the practice, though being fairly common or widespread, is far from being the predominant substance-abuse problem in most areas. A survey conducted in New York City and Chicago found that the average age of persons inhaling volatile solvents was 14.8 years, with a range of 10 to 17. However, users as young as 7 and 8 have been reported in such surveys. There is a trend in larger urban areas towards users being younger at the initial experience than they were in the past, and being younger than their counterparts in rural settings. The mean age for solvent experimenting has been reported to be 2 years younger than the average age for first alcohol use.

Males are much more likely to use inhalants than are females, often by ratios as large as 10 to 1. In general, inhalant users are found in small groups within larger populations. These persons usually share common social, economic, or cultural backgrounds. Survey data suggest that Blacks are infrequent solvent users, and that the problem is more frequent among Hispanic Americans, Native Americans, and White Americans of Appalachian extraction. Poor education, lack of social skills, and economic deprivation seem to be positively correlated with chronic solvent abuse in these populations. Solvent sniffers are often from families that are disorganized, or lack an effective father figure. Solvent sniffers tend to exhibit emotional outbursts, passive aggressive tendencies, antisocial behavior, social inadequacy, impaired judgement, and bashtfulness. These are characteristics commonly found in alcoholics [B*Korman, 1977].

Although solvent use is generally thought to be a practice of young people, there is some use by adults as well. Adult use is usually associated with specific substances and groups, such as incarcerated or institutionalized persons using gasoline, or health-care professionals using anesthetics. In general, however, chronic adult solvent users are persons who began using these substances in adolescence and have failed to stop use as they grew older. A high incidence of psychological and physical pathology is found in the adult chronic sniffer group.

There are two major groups of volatile substance users: experimenters who use these substances on a temporary basis before moving on to other substances, and chronic abusers. Available evidence seems to indicate that the most common pattern of solvent sniffing is the occasional type, in which use is aimed at getting "high," and is practiced in a group setting. Inhalant experimentation seems to be fairly widespread among young people with "almost everyone trying it." Among the general population, however, abuse is rare; chronic abuse is a phenomenon primarily among the poor and young. Children usually start sniffing with

their peers. Often groups involved with inhalant use develop their own systems for obtaining and distributing the "agents of choice." These groups develop in certain neighborhoods, and not in others. Usually inhalant abusers do not develop their own language or rituals, and are not considered to be part of the drug subculture.

COMMENTS : Solvents & Aerosols

CHEMISTRY & PHARMACOLOGY

Chemical constituents of many of the classes of products used by inhalant sniffers to get high are mixtures of two or more volatile substances. Although the specific chemical composition of these products varies remarkably from product to product, the subjective effects resulting from inhalation are remarkably similar. When the clinician is dealing with the side effects or toxicities of a specific product, it is important to know the specific ingredients of the product in question, because certain pharmacologic or toxic effects may be manifested by single ingredients independently. In addition, it has been reported that individual ingredients of certain products may act in an additive or synergistic fashion in producing psychoactive effects of toxicities. It should be emphasized that a manufacturer may change ingredients in a product without warning or notice and, therefore, a person using a particular commercial product may inadvertently be exposed to toxins simply because the formula has been changed. The previously "safe" product has thus become toxic after the addition or substitution of one or more ingredients. With the exception of the anesthetics, most of the commonly used substances are compounds with chemical constituents from different groups, that is, aromatic or aliphatic hydrocarbons, alcohols, ketones, esters, and halogenated hydrocarbons (fluorinated compounds and di- and trichlorinated compounds).

Substances that are sniffed for their psychoactive effects are highly lipid soluble, by nature of their chemical structure. Thus, they are rapidly transferred across the membranes of the lungs into the bloodstream and thence to the brain. The inhalation of solvents or aerosol substances is, therefore, a quick and easy way to deliver intoxicants to the brain in concentrations that will produce altered states of consciousness. Because these substances are lipophilic, they tend to become concentrated in body tissues that have high lipid content, such as the CNS or fat tissue. It is presumed that the effect of these substances on consciousness is related to their lipid solubility, which causes CNS depression by impairing membrane permeability and neuronal transmission. In addition, high lipid solubility means that the substances are taken up and stored by body lipids until they are excreted or metabolized. Normally there is an equilibrium between the substance stored in its lipid "depot" and the substance in the blood. The substance in the blood is usually excreted by exhalation. Further, these substances are metabolized via the liver and the resulting products are excreted in the urine. However,

because these substances are stored in lipid depots and then slowly eliminated or metabolized, there may be a cumulative effect leading to toxicities if an additional substance is inhaled before that stored in the tissues is eliminated.

METHODS OF ADMINISTRATION

For the most part, the substances to be inhaled are placed in a closed container, such as a plastic or paper bag. Liquids may be poured on a rag, whereas glue is usually squeezed into the bag directly from the tube. The opening of the bag is held tightly around the nose and mouth, and the vapors are inhaled until the desired effect has been produced, or until all of the vapors have dissipated. In some cases, fumes are inhaled from the original container, or a saturated rag is simply held in the cupped hands. Small tubes of model airplane cement, because of their size, are handy containers.

People who use aerosol propellants normally separate the propellant from other can contents. A variety of methods have been developed, such as holding the can in an inverted position to release the gas propellant only; "filtering" the contents through a rag; spraying the contents into a bag and inhaling the vapors only; or inflating a balloon. A few users may even resort to spraying the contents of an aerosol can directly into the nose or mouth.

In many instances, the substance to be inhaled is heated or warmed to enhance vaporization. The sniffer may warm the bag in the cupped hands, or hold it over a radiator or hot plate. In other situations, the substance may be placed in a warm pan to facilitate vaporization. It has been estimated that various warming techniques may increase the concentration of the inhaled substance up to 50 or more times over the allowable industrial concentration.

Sniffers may use their own bags, or they may share them in social settings with other users. The bag, in addition to being an effective way of self-administering concentrated vapors, may be a "safety" device, because as a person inhales these substances to the point of unconsciousness, the grip on the bag relaxes, allowing it to fall away from the mouth. This effectively limits the exposure and protects the user from prolonged respiratory depression, anoxia and, perhaps, death. In this regard, use of plastic bags is more dangerous, because the seal formed between the facial skin and the plastic is much more efficient. With the exception of sudden sniffing deaths from inhaled aerosol propellants (mentioned later), suffocation from using plastic bags is the leading cause of death for sniffers.

CLINICAL EFFECTS

The primary pharmacologic effect of substances used by inhalant abusers is general CNS depression, producing an altered state of consciousness, which resembles general anesthesia. Inhalant use leads to a dose-dependent depression of the CNS. With increasing dose, one might experience effects ranging from mild intoxication or excitation, to total loss of consciousness. In general, use of solvents leads to disinhibition euphoria (loss of inhibitory controls and a subjective "high") which is common to the use of all CNS depressants. The effects are immediate, beginning within one or two breaths, lasting for the duration of sniffing, and ending gradually after inhalation ceases. Some effects, for example, a faint odor, may linger long after the high is gone. The high is often followed by sedation or a feeling of drowsiness, which may last one or more hours. Recovery from the effects of solvent sniffing may also be accompanied by nausea and headache. Use of inhalants, depending upon dose and depth of intoxication, is often associated with partial or total amnesia for the period of intoxication. This may be a drug-state dependent learning effect.

A number of adverse side effects have been experienced during and after use and inhalation. These include coughing and sneezing, chest pain, irritation of the nasal passages and bronchial tree, nausea, vomiting, diarrhea, irritation of the eyes, visual disturbances, and vague muscle or joint pain. Anorexia is common in users, but perforation or ulceration of the nasal mucosa have not been reported. These effects appear to be transient and largely reversible. Chronic toxicity resulting from self-administration of inhalants is discussed in a later section.

Following inhalation, a number of transient effects may be experienced. These effects may range from simple drowsiness or lightheadedness to delusions of omnipotence or unusual strength to full-blown auditory or visual hallucinations. The usual responses are giddiness, slurred speech, ataxia, impaired judgment, and a marked sense of euphoria. The person may act and appear outwardly as though "drunk" on alcohol. Users often report, however, that the degree of euphoria is more pronounced than that experienced with alcohol (probably because of the rapidity of absorption and distribution to the brain). While under the influence of volatile inhalants, the user may do harm to him- or herself or to others, owing to feelings such as indestructibility or being able to fly.

Chronic use of these compounds may lead to psychologic dependence and tolerance, but there is little to suggest that people become physically dependent on solvents and aerosols. Habituation occurs regularly, and users pick a favorite product to use for their chemical respite from reality. Alternate substances are frequently employed if the favorite is unobtainable. Compulsive chronic use has been reported.

TOLERANCE & DEFENDENCE

Tolerance to the CNS effects of these products is experienced by sniffers who use them regularly for long periods of time. The length of time for tolerance to develop is variable, and data on cross-tolerance are not available. A few persons have experienced vague symptoms upon cessation of solvent use, but these have been attributed to psychologic rather than to physical causes [B*Bowman, 1977]. Physical dependence, if it occurs, is rare, and no distinct abstinence syndrome has been identified.

TOXICOLOGY

Current data suggest that, in general, toxic effects of volatile inhalant use are minimal in the occasional user. An exception is the "sudden sniffing death" associated with aerosol exposure. Apart from deaths caused by plastic bag suffocation, falling from heights, accidental explosions, or automobile accidents, the primary dangers are damage to the CNS, the peripheral nerves, the kidneys, the liver, and the bone marrow. These injuries may require a considerable period of time for recovery if, indeed, they are reversable at all.

The toxic effects of sniffing most volatile substances are generally believed to be transient in nature, that is, they are related to the concentration of the substance in the body. They cease once excretion brings the body concentration below minimum toxic levels. There are a few substances, however, that present serious health hazards. Effects of a transient nature include an acute organic brain syndrome characterized by confusion, dizziness, inability to concentrate, disturbances in gait, and loss of memory. Beyond these problems, which are common to most of the substances in this category, medical and toxic symptoms associated with substance inhalation are related to the specific chemical ingredients of the substances used.

Those ingredients commonly found in commercial products that cause toxic problems include benzene, tetraethyl lead, carbon tetrachloride, n-hexane, and halogenated hydrocarbons, found in aerosols. Much of the toxicity data available on ingredients of abused solvents are derived from industrial, occupational, and laboratory studies, rather than from the clinical picture of chronic inhalation of psychoactive substances with hedonistic intent.

Halogenated Hydrocarbons

The most serious toxicity problem and threat to health associated with inhalation of substances is the so-called "sudden sniffing death" [B*Aviado, 1977]. This problem is most often associated with inhalation of fluorocarbon aerosol propellants. Some aerosol containers now contain nontoxic, nonfluorocarbon propellants. Thus, these are not associated with this health hazard. Recent Federal regulations limiting fluorocarbons for

this purpose and because of environmental concerns have reduced the likelihood of sudden sniffing deaths, but have not eliminated it.

Halogenated hydrocarbons sensitize the myocardium to catecholamine-induced arrhythmias. This phenomenon is well known to medical personnel who administer halogenated hydrocarbon anesthetic compounds for surgical anesthesia. Bass [J*1970] reported over 100 sudden sniffing deaths. In 6 eyewitness accounts, the victims were reported to have become wild and hysterical, or to have run about 100 feet prior to sudden death. Release of epinephrine from the adrenal gland, caused by stress, excitement, or anoxia, has been associated with the phenomenon. The released epinephrine, a strong cardiac stimulant, may then precipitate fatal ventricular arrhythmias (fibrillation, arrest) in the presence of a sensitized myocardium.

The Consumer Product Safety Commission has compiled accounts of more than 300 deaths relating to the intentional inhalation of aerosol sprays. The Commission has stated that death associated with intentional inhalation of aerosols is a great hazard of packaging products in aerosol containers.

Trichloroethylene can also produce cardiac arrhythmias, acute renal failure, hematuria, myocarditis, and heart failure. Inhalation of Carbona (which contains trichloroethylene and trichloroethane) has been associated with hepatic damage. Carbon tetrachloride, chloroform, and ethylene dichloride are toxic compounds, and cause CNS edema, pulmonary edema, damage to the heart, spleen, and liver, and cardiac arrhythmias as well [B*Aviado, 1977].

Aliphatic Hydrocarbons

Reports have been scanty of organic disease resulting from the inhalation of many of the aliphatic hydrocarbons. In some cases, adequate documentation of hematological, urinary, and chemical determinations is missing and, therefore, potential toxicity may have been overlooked. Most of the petroleum distillates have been associated with little toxicity even when they have been inhaled to the point of hallucinosis or stupor. Exceptions are products containing n-hexane and gasoline.

N-hexane is a volatile solvent often found in certain types of model airplane cement. Chronic inhalation of a glue containing n-hexane has produced pain, tingling, and weakness in the legs [B*Bruckner & Peterson, 1977]. These neurologic deficits may last a year or longer.

Gasoline, in addition to causing deaths from explosions, has two potentially hazardous ingredients. Excessive exposure to gasoline fumes has caused chronic pulmonary irritation, anemia, and neurologic problems. (ii) Gasoline contains enough benzene to be hazardous. Benzene is harmful to the formed elements in the

blood, and has been reported to cause chromosomal damage and leukemias in humans [J*Vigliani & Forni, 1976]. In addition, some types of gasoline contain the "antiknock" ingredient, tetraethyl lead, "Ethyl." Persons who inhale leaded gasoline, therefore, may experience symptoms of systemic lead poisoning. Permanent damage to the CNS has been known to follow inhalation of large quantities of tetraethyl lead. [J*Law & Nelson, 1968] reported one case of toxic psychosis from lead encephalopathy in an adult sniffer.

Aromatic Hydrocarbons

As previously mentioned, benzene is a highly toxic substance. High concentrations of any aromatic hydrocarbon, of course, can cause coma and death, but benzene is also known to cause bone marrow aplasia, anemia, and necrosis, and fatty degeneration of the liver and heart. Toluene is less toxic than benzene. Although industrial exposure to toluene has been reported to cause toxicities, it is likely that many of these problems were caused by benzene contamination. In more recent times, toluene has been supplied in purified form and few toxic symptoms have been reported with its use. Liver and kidney damage have been absent in animals given toluene, although there have been scattered reports of blood dyscrasias and anemias, or CNS damage, in animals and humans. Xylene has essentially no toxic effect on bone marrow, and is minimally toxic to other organs. However, inhalation of xylene vapor is irritating to mucous membranes [B*Bruckner & Peterson, 1977].

Ketones & Esters

Acetone, methyl ethyl ketone, and other ketones have been reported to cause dermatitis, numbness of the extremities, irritation of mucous membranes, and CNS depression, but rarely any other toxicities. Although acetone exposure with cutaneous absorption has been reported to cause intoxication, there have been no reports of significant toxicity from intentional hedonistic use of products containing solely acetone. Use of esters (ethyl acetate, amyl acetate, butyl acetate) is not known to cause liver, kidney, or hematologic damage [B*Couri & Nachtman, 1977].

COMMENTS: Nitrous oxide

Nitrous oxide was discovered in the late eighteenth century. In fact, the first documentation of its psychoactive use was in 1799, when the 21-year-old British chemist, Humphrey Davy, inhaled the gas, originally for scientific purposes. "Nothing exists but thoughts," he observed, and proceeded to share the gas with his friends. Laughing-gas parties and demonstrations soon became the social event of the "beautiful people" of the nineteenth century. Nitrous oxide peddlers became a popular attraction of traveling medicine shows.

Nitrous oxide use has continued among certain groups, although it no longer enjoys the social standing it once had. Today, its use is confined to dentists and other health professionals who use medical-grade nitrous oxide, which is manufactured for use as a propellant in whipped cream dispensers.

CHEMISTRY & PHARMACOLOGY

Nitrous oxide is a colorless gas without appreciable odor or taste. Under pressure in containers it is a liquid, but it returns to a gas upon release. It is heavier than air, and is the only inorganic gas that is practical for use as an anesthetic. Nitrous oxide has a relatively low solubility in blood. It does not combine with hemoglobin. In the blood it is simply in solution. Nitrous oxide is excreted unchanged, primarily via the lungs. Because nitrous oxide is not decomposed, the oxygen in nitrous oxide is unavailable to support tissue respiration. Nitrous oxide will support combustion, although it will not explode.

Nitrous oxide is a general anesthetic and analgesic. When used alone, however, it is not a good anesthetic because it is not effective in producing deep surgical anesthesia, and it is not a good muscle relaxant. Its primary disadvantage as an anesthetic is that in order to produce effective anesthesia, one must increase the concentration to nearly 100% and, thus, increase the danger of asphyxiation. It is necessary to administer oxygen at 35% or more in order to prevent anoxic damage during nitrous oxide anesthesia.

Nitrous oxide is rapidly transferred across alveolar membranes. It enters the pulmonary capillaries more rapidly than oxygen. It is rapidly excreted upon termination of inhalation, and the user recovers in a short period of time. Inhalation of nitrous oxide produces a brief high. The effects begin almost immediately upon inhalation and lasts only a few minutes before the gas is blown off in expired air. If the gas is inhaled for a longer period of time, there is a longer high. The high is accompanied by amnesia and analgesia. Occasionally a person may experience delirium, hallucinations, nausea, or vomiting, but recovery from the substance is usually devoid of serious sequelae.

PATTERNS OF USE

Nitrous oxide comes in two kinds of containers. The first consists of the large tanks used for medical and dental purposes of anesthesia. Pure, medical-quality nitrous oxide is not available to the general public. In fact, most suppliers of nitrous oxide for medical purposes will only sell it to legitimate medical facilities, requiring identification and often a controlled-substances license number. Most people who use medical-grade nitrous oxide are dentists, physicians, medical students, or hospital personnel who sniff it occasionally or on a regular

basis. Use is usually limited to those persons who have ready access to the gas. However, these nitrous oxide tanks are frequently the target of thieves, who search for nitrous oxide on hospital loading docks. Most hospitals now keep these tanks in locked rooms.

The second way of obtaining nitrous oxide for abuse is in small cartridges or torpedo-shaped cylinders, which are intended for use as propellants for whipped cream dispensers. Although these cylinders are marketed under various names by different companies, they are known generically by the most famous name associated with these products: "Whippets." Nitrous oxide in Whippets is not intended for human consumption and, as such, is not governed by the stringent regulations regarding purity that cover medical nitrous oxide. Therefore, Whippets may be sold to the public, and have become popular items in "head shops." One such shop in Cincinnati sells thousands of units of Whippets each weekend.

Methods of using nitrous oxide vary with the type of container. Medical personnel may inhale it directly from an anesthetic mask. Persons using nitrous oxide from purloined cylinders may substitute a gas mask, obtained from army surplus stores. Whippet users, on the other hand, use a device that punctures the end of the cartridge, releasing the gas into a balloon. The gas is then inhaled directly from the balloon. Persons at rock concerts may be observed to share their laughing gas with friends by passing the balloon from person to person in much the same way that groups share a joint. An alternate way of using Whippets is by inhalation from a device that releases the gas through an oxygen-type mask. Nitrous oxide abusers have also been reported to inhale the substance from aerosol whipped cream cans normally found in supermarkets. In these cases, the can is tilted so that the whipped cream remains in the can and only the gas escapes. The gas is inhaled through the mouth directly from the can.

TOXICITY

Apart from the major consequences of hypoxia and asphyxiation, few toxic problems have been associated with nitrous oxide. Two nitrous oxide-related deaths have been attributed to asphyxiation. In Cincinnati in 1981 there was a death related to nitrous oxide abuse. This particular person was breathing 100% nitrous oxide through a gas mask attached to a cylinder of nitrous oxide. When found by his mother, he was unconscious, and was pronounced dead on arrival at a local hospital. Of course, nitrous oxide inhalation without sufficient oxygen may cause permanent nerve damage if the period of hypoxia is of sufficient duration [J*Helisten, 1975].

The effects of nitrous oxide on the liver and kidneys are usually assumed to be negligible. In the absence of hypoxia, there are few changes in other visceral organs. Although there have been reports of bone marrow depression and death following prolonged exposure to nitrous oxide, the primary toxic problem

associated with recreational use of this gas is peripheral neuropathy. Gutman et al. reported a case involving progressive paresthesia involving both legs in a dentist abusing nitrous oxide. In 1978, Layzer et al. reported neuropathy in three health professionals who abused nitrous oxide. Sahenk reported polyneuropathy in a 23-year-old woman who was using as many as 72 Whippet cartridges per night. An interesting note in the latter case is that the analysis of the Whippets revealed the presence of 26 foreign compounds in addition to the nitrous oxide. Three of these (trichloroethylene, toluene, and phenol) are neurotoxins. Most of these persons improved following discontinuation of nitrous oxide use. There have been reports that parenteral vitamin B₁₂ may improve the nitrous oxide-induced neuropathy, because nitrous oxide apparently inactivated vitamin B₁₂ [Deacon et al., 1978].

Nitrous oxide may also cause some organic or psychologic mental dysfunction in man and animals. [J*Brodsky & Zuniga, 1975] also reported on a 32-year-old man who developed psychotic and toxic-organic features after 6 months of almost daily inhalation of nitrous oxide. Because nitrous oxide use may impair reaction time and judgement, persons under the influence may become involved in fatal accidents while driving or if engaged in hazardous occupations. Nitrous oxide has also been reported to contribute to infertility, abortion, fetal malformations, and cancer.

COMMENTS: Ethyl Chloride

Use of ethyl chloride is becoming fashionable in certain areas of the country. In Cincinnati, a local high school guidance counselor has reported use, and we have heard of use in discos, particularly in New York. Although ethyl chloride was formerly used as a general anesthetic, its use is now largely restricted to topical application as a local anesthetic. For this purpose it is sprayed on an area of the body, and rapid evaporative cooling deadens sensations in the same way that cooling with ice does. If, however, the vapors of the drug are inhaled, they cause general anesthesia, stupor, and unconsciousness. In lower concentrations, ethyl chloride causes a loss of coordination, dizziness, confusion, an alcohol-like intoxication, and weak analgesia.

Use of ethyl chloride is not without hazard. Exposure to low concentrations may produce nausea, vomiting, cough, headache, perspiration, salivation, and tremor. The more serious adverse effects include sudden circulatory and respiratory failure, disorders of heart rhythm, and liver damage.

Ethyl chloride is restricted to sale on prescription only. It is packaged in spray bottles or cans. For inhalational use, it must be placed in a closed container and allowed to evaporate so that the vapors may be inhaled.

COMMENTS: Volatile Nitrites

HISTORY & LEGAL STATUS

Amyl nitrite has been used medically since 1867 to provide relief from attacks of angina pectoris. It is packaged in small, thin glass capsules covered by cloth webbing; these are then crushed between the fingers to release fumes, which are inhaled during anginal pain. In the late 1960s, sales of amyl nitrite rose sharply due to its nonmedical use as an agent for getting high, and as an aphrodisiac. It acquired the street name "poppers" because of the sound created by crushing the capsules.

The widespread use of volatile nitrites is a fairly recent phenomenon. These compounds have been used for some time by male homosexuals (B*Bruckner & Peterson, 1977). During an early period of use (the 1950s and 1960s), some pharmacies sold amyl nitrite "over-the-counter" to people who would present themselves as patients suffering from angina pectoris. In the late 1960s, the misuse of volatile nitrites became widespread. Amyl nitrite was classified as a prescription drug in 1968. In the early 1970s, the United States witnessed the emergence of a multimillion dollar business in marketing various volatile nitrite compounds (see Table 1-4 for a partial list of such products and their contents). Because these products are marketed as "room odorizers," they do not fall under the regulatory jurisdiction of the FDA. Thus, they are not required to meet FDA standards for drug safety and effectiveness. Rather, the products are under the regulatory jurisdiction of the Consumer Product Safety Commission, and only need meet the same safety standards of products such as Air-Wick [R]. There is pressure from some legislators to change the laws so that such products would become illegal without a prescription. It is difficult to predict the impact that such laws would have on the use of volatile nitrites.

PATTERNS OF USE

The usual patterns of use involve inhaling the vapors directly from an opened bottle or crushed "popper," or from a variety of inhalers into which the nitrite solution has been transferred. The vapors are inhaled once or several times during an episode of use. The snorting may be done just to get high, as a prelude to sexual activity, or just prior to orgasm.

Volatile nitrites are psychoactive agents that have the reputation for being aphrodisiacs. They have been and continue to be popular among the male homosexual population. In fact, within some male homosexual groups, the use of these compounds has become a requirement for group membership. Although male homosexuals represent a sizable number of the total population of volatile nitrite users, the use of volatile nitrites is by no means confined to homosexuals. Some bisexuals, heterosexuals, and female homosexuals also enjoy the use of these compounds. In addition, some inhale volatile nitrites simply as a social drug for

Table 1-4: Contents of Various Nitrite Preparations

	Nitrite	N-butyl alcohol	Isoamyl alcohol	Isobutyl alcohol	Butyl nitrite	Amyl nitrite
Rush	+			+	+	
Toilet water	+			+	+	
Heart-on	+			+	+	
Locker room	+			+	+	
Ban Apple Gas	+	+			+	
Bullet	+		+		+	
"Street poppers" (Cincinnati)	+		+			
"Street poppers" (Dayton)	+	+				
Rocket	+				+	
Bolt	+				+	
Crypt Tonight	+				+	
Joc Aroma	+				+	

relaxation, for facilitation of a sense of abandon while dancing, or for solo or group inhaling just to get high.

SUBJECTIVE EFFECTS

The use of volatile nitrites has been reported to aid in intromission when used by male homosexuals during anal intercourse [J*Everett, 1972; J*Gay & Sheppard, 1972; J*Hollister, 1975]. Although the mechanism is unknown, several of the drug's actions may contribute to this. Volatile nitrites relax intestinal and rectal smooth muscle, as well as the internal anal sphincter. It has also been suggested that inhalation of volatile nitrites may relax the external anal sphincter, which is composed of striated muscle and is under voluntary control, by causing general disinhibition and relaxation, or perhaps through transient cerebral ischemia that accompanies the hypotension.

Sensations of heightened intensity and increased duration of orgasm have been reported by persons who inhale volatile nitrites in conjunction with sexual activity. The authors speculate that a reflex sympathetic nervous discharge, mediated by baroreceptor responses to hypotension (secondary to vasodilation) induced by inhaling poppers, may add to the sympathetic discharge of orgasm to produce indeed the sensation of a "longer-stronger-deeper" and "more powerful" orgasm.

It is interesting to compare different users' perceptions of the actions and effects produced by volatile nitrite compounds. Most patients with angina pectoris who have been treated with amyl nitrite complain about the smell, headaches, and general discomfort. The drug is not perceived by these persons as producing a high. In short, they do not like it. Medical students who have used these compounds to dilate retinal blood vessels while learning to use an ophthalmoscope complain about dizziness and the obnoxious smell. However, street users (in whom the drug presumably has the same actions) say they like the high and the rush it produces! This is yet another example of the influence of the psychological and social levels of the bio-psycho-social model on the interpretation of a drug's action at the Biological level.

CLINICAL EFFECTS

The recent upswing in the use of volatile nitrites is an interesting phenomenon. With the exception of significant adverse effects and death that can occur in acute overdose, the volatile nitrites seem relatively harmless in short-term, low-dose use. The compounds have not been in widespread use long enough to determine the nature, if any, of their chronic toxicity. Recall, for example, that the increased incidence of lung cancer following cigarette smoking does not occur for 10 years after significant exposure to cigarette smoke.

The pleasurable sensations that are perceived following inhalation of the volatile nitrites (like those produced by other chemical agents used to affect mood, perception, thought, or behavior) result from the combined effect of the pharmacology of the specific agent, the person's expectations, the environment, and the social setting in which they are used. In this regard, a set of beliefs about the actions, uses, and effects of volatile nitrites that has evolved with input from advertising and users, is necessary for continued use of these agents. For without this conditioning, the naive person exposed to such chemicals usually finds them obnoxious and irritating.

The volatile nitrites are smooth muscle relaxants, and therefore are arteriolar vasodilators. When these agents are inhaled, the effects begin with one circulation time, and most of the actions terminate within 2 minutes. A number of systems in the body are affected, and the following effects are observed:

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Cardiovascular effects

Profound vasodilation, resulting in hypotension and cutaneous flushing are followed by reflex vasoconstriction, tachycardia (due to sympathetic discharge), and transient EKG changes (e.g., inverted T waves and depressed ST segments). The actions of the usually consumed doses of volatile nitrites are more pronounced and short-lived than the effects of the usual therapeutic dose of nitroglycerin.

CNS effects

These can include light-headedness, weakness, nausea, ataxia, delirium, headache (short-lived "nitrite headaches" or prolonged pulsatile headaches), syncope (following profound vasodilation), tolerance, sedation, and anesthesia.

Hematologic effects

Although methemoglobinemia has been documented after oral and parenteral administration (J*Levria, 1970), and has been repeatedly mentioned as a possibility after inhalation, clinically significant methemoglobinemia is uncommon with the hedonistic use of these substances. Normocytic, normochromic anemia has been reported after industrial exposure.

Ocular effects

Supraorbital pain may occur. Claims in the older literature of increased intraocular pressure have been refuted by recent evidence.

Pulmonary effects

Irritation of the lungs, sinuses, and nasal passages can occur. Physicians in the San Francisco Bay area report multiple cases of inflammatory sinusitis secondary to pouring the liquid down the nose.

There is little information about the doses and duration of exposure necessary to produce the more serious adverse effects. Most of the toxicology literature on butyl nitrite and amyl and butyl alcohol refers to concentrations in the air breathed by industrial workers.

A single case of death from isobutyl nitrite abuse has been reported. A 30-year-old man died from cardiac arrest after drinking an isobutyl nitrite product. The oral ingestion of volatile nitrite products is a dangerous practice because the user cannot titrate the dose as well as when it is inhaled.

Research regarding the n-nitrosylation reaction raises the issue that repeated use of these products could increase the risk

of cancer because inhaled nitrites could react freely with endogenous trivalent nitrogen compounds to produce nitrosamines. Some nitrosamines are known to be carcinogens. It remains to be determined whether or not nitrosamines are formed when these preparations are inhaled, and if so, are they carcinogenic? In addition, it remains to be determined just what amount of nitrite exposure by inhalation is required to produce nitrosamines in sufficient amounts to increase cancer risk.

DRUG-INTERACTIONS

COMMENTS: Drug Interactions

Drugs interact with other drugs, that is, alter the nature of their effects by virtue of certain basic phenomena. These can be classified in terms of:

1. direct effects (positive or negative) on common sites of action;
2. indirect effects on a common system or function;
3. effects on transit of drug to its site of action;
4. effects on drug metabolism and elimination.

The actions of many prescription, street, and over-the-counter drugs can be modified by alcohol, and other drugs [B*Cadwallader, 1979]. The first clue to the occurrence of a drug-drug interaction may be an unexpected response to the therapeutic administration of a drug. For this reason, health professionals should be knowledgeable about the common interactions involving drugs of abuse, both licit and illicit. Serum (total) drug levels are misleading when multiple drugs are used [R*SPIT].

Drug interactions, especially those involving street drugs, are not always predictable. Street drugs vary widely in actual qualitative and quantitative content. Even extremely well-documented drug-drug interactions may rarely occur and/or prove clinically significant in only a small percentage of people taking the drug combination. All of the variables that affect drug actions also modify drug interactions. These include:

1. the drug;
2. its dosage, interval, duration & route;
3. its additives, including other drugs, foods, fillers, dyes, parenteral fluids, diluents, containers or coatings;
4. chemical or pharmacological interactions between admixtures of drugs;
5. personal factors contributing to variability in response to drug such as: age, race, sex, weight, genetic factors, health status, pharmacokinetics, acquired or intrinsic tolerance, idiosyncratic/immune susceptibility, temperature, altitude, set and setting, diet & nutritional factors, time of day, relationship

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with prescribing physician or drug pusher, awareness of therapeutic or intended actions, health beliefs and optimism/pessimism about drug safety and efficacy;

6. factors which cause altered therapeutic response such as errors in administration, false lab results, toxicity, drug incompatibility, concurrent illnesses which interact with the drug.

Drug interactions are not all intrinsically adverse; in fact, some drug interactions are clinically useful.

It is important to know the quantities of drugs being taken, as well as the length of time over which the person has been using each of the drugs (pattern of use). Only with this information can one determine whether the drug use is acute, chronic, or being withdrawn. Because patients may be in any phase of drug use (acute, chronic, or withdrawal), it is important to realize that specific sets of symptoms (such as sleeplessness, and excessive anxiety) can be produced by the acute use of some drugs (e.g., stimulants) or withdrawal of other drugs (e.g., sedative-hypnotics). How a second drug might interact with the first drug also depends on how the second drug is used, that is, acutely, chronically, or in withdrawal. The variables of acute and chronic use and withdrawal are diagrammed in Table 1-5, which shows the possible conditions under which two drugs (drug A and drug B) might interact. Timing and dose are important variables in predicting, explaining, and modifying drug actions.

Table 1-5: Drug administration conditions for two interacting drugs

	ACUTE USE DRUG 2	CHRONIC USE DRUG 2	WITHDRAWAL DRUG 2
ACUTE USE DRUG 1	1a/2a	1a/2c	1a/2w
CHRONIC USE DRUG 1	1c/2a	1c/2c	1c/2w
WITHDRAWAL DRUG 1	1w/2a	1w/2c	1w/2w

COMMENTS: Drug-Alcohol Interactions

Alcohol is no exception to the general rule, for it interacts extensively with a wide variety of drugs [J*Anonymous, Medical Letter, 1981b; J*Weller & Preskorn, 1984]. In fact, the additive CNS depression effect of alcohol with sedative-hypnotics, and other CNS depressants is responsible for the greatest number of drug-related overdose deaths in our nation today.

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Chronic alcoholism is a multisystem disease that affects the metabolic capacities of most organs, including the liver, brain, and kidneys. Early in the course of the disease, liver enzyme synthesis is usually induced, which increases the liver's capacity to metabolize a wide variety of drugs [J*Anonymous, Medical Letter, 1981b]. As the disease progresses and more hepatic tissue is destroyed, the liver loses its capacity to perform essential metabolic tasks, such as the synthesis of urea from ammonia. In the condition of prehepatic encephalopathy, the administration of some drugs, for example, meperidine, can precipitate hepatic encephalopathy. Hypoalbuminemia, which often accompanies severe toxic liver injury, can dramatically affect the metabolism, distribution, and elimination of a wide variety of drugs that bind to plasma albumin--for example, chloral hydrate. Late in the disease of alcoholism, the liver's ability to synthesize vitamin K dependent clotting factors is compromised. Decreases in renal function interfere with the elimination of drugs and their metabolites that are normally eliminated via the urine. The brain gradually loses its capacity to compensate for the presence of alcohol, presumably due to the destruction of nervous tissue by alcohol. Thus, early in the course of alcoholism, the brain may exhibit profound cross-tolerance to other sedative-hypnotics. However, as alcoholism progresses, the brain's capacity to become tolerant and cross-tolerant to other drugs decreases (see Table 1-6).

Table 1-6: Progressive Results of Alcohol Use on Body Drug Metabolism

	EARLY OR ACUTE USE	INTERMEDIATE USE	LATE USE
Drug metabolic capacity	Slowed drug metabolism	Accelerated drug metabolism	Intermediate drug metabolism til hepatic failure
Kidneys	Healthy, ADH responsive	Weakened ADH response	Renal failure
Brain	Brisk drug effect	Tolerance	Loss of tolerance
Liver	Damaged by alcohol	Enzyme induction	Considerable damage

Some other drug interactions involving alcohol include:

1. Isoniazid (INH) action is decreased with chronic alcohol use;
2. Phenformin (DBI) causes lactic acidosis when combined with alcohol;
3. Salicylates cause or exacerbate gastrointestinal bleeding;
4. Tolbutamide's antidiabetic effects are potentiated by

acute administration of alcohol, but are antagonized when combined with alcohol chronically;

5. Antihistamines, benzodiazepines, major tranquilizers, meprobamate, and narcotics potentiate the CNS depressive effect of ethanol;
6. Disulfiram (Antabuse), chloramphenicol (Chloromycetin), chlorpropamide (Diabinese), furasolidine (Furoxone), griseofulvin (Fulvicin), metronidazole (Flagyl), phen-tolamine (Regitine), quinacrine (Atabrine), tolbutamide (Orinase) all cause a disulfiram-type reaction, although the last eight drugs listed produce only a mild reaction when one occurs at all;
7. Barbiturates, phenytoin (Dilantin [R]), sedatives, major and minor tranquilizers, tolbutamide (Orinase), and warfarin-like anticoagulants are potentiated during acute alcohol intoxication, but inhibited after chronic alcohol abuse.

Antagonists

Discovery of a specific ethanol antagonist has long been the hope of many physicians (as well as alcoholics). However, to this day we have developed no effective amethystic, or sobering agent. It has been proposed that caffeine (coffee) and other drugs that excite the CNS by very different means might indirectly antagonize the toxic depression due to alcohol, but none have proven efficacious and reliable. Analeptics, such as pentylenetetrazol, are especially dangerous where the potential for alcohol withdrawal seizures exists. Naloxone has been shown to inhibit dependence to ethanol and morphine to suppress alcohol withdrawal in mice [J*Blum, Wallace & Futterman, 1977; J*Blum et al., 1976].

Alcohol & Amphetamines

Amphetamines are not effective antagonists to large doses of alcohol. Neither amphetamines nor caffeine are effective at "sobering up" someone who is drunk [J*Newman & Newman, 1956]. In certain persons, alcohol promotes hostility and violent acts. The result of such a combination is a wide-awake drunk. In all persons thus far tested, large doses of amphetamines caused paranoia, hostility, and violent behavior. The behavior usually changes gradually, over a period of days to weeks when the dose of amphetamines is sufficient. The combined chronic use of alcohol and amphetamines is responsible for a great deal of the senseless violent behavior in our society. Acute and chronic use of amphetamines and the chronic use of alcohol tend to elevate blood pressure. Chronic use of amphetamines alone in high doses results in the development of paranoid psychosis. Alcohol, consumed alone, can produce other psychiatric conditions, namely, Wernicke's encephalopathy and Korsakoff's syndrome. The amphetamine-induced changes in mentation are particularly dangerous when combined with those changes brought on by chronic alcohol abuse.

Potentiation

Synergy of action can be anticipated as a direct common mode of interaction between volatile anesthetics (employed for general surgery) and alcohol. The inhalational agents are thought to disrupt the function of excitable membranes in a fashion much like that of alcohol. Thus, if an intoxicated accident victim appears at the emergency service requiring surgery, it is very likely that he or she will need less anesthetic to achieve a surgical plane of anesthesia. However, the case is considerably more demanding when dealing with the apparently sober alcoholic patient, for he or she will require more anesthetic than would seem appropriate because of the tolerant condition. Lethal levels of depression do not rise appreciably with tolerance, and so the margin of safety is dangerously narrowed in such cases.

An instance of indirect additive interaction is provided by the toxic effect of ethanol on the gastric mucosa. Other agents, like aspirin (which might be taken for hangover headaches), exacerbate the gastritis in concert with alcohol.

Because alcohol is consumed orally, transit to its site of action begins in the GI tract and is thus influenced by drugs that alter gut secretion and motility. Antimuscarinic agents will retard motility and thereby delay stomach emptying. Because ethanol absorption proceeds more rapidly in the intestine than in the stomach, the net effect will be a slower total rate of uptake into the vascular compartment. Once in the blood, alcohol distributes itself readily to well-perfused tissues. Naturally, if cerebral perfusion is altered, the change will have a direct effect on the rate and intensity of ethanol induced CNS depression.

Alcohol & Anxiolytic Sedatives

When taken concomitantly, alcohol and anxiolytic sedatives increase the CNS depression caused by alcohol [J*Linnoila, Saario & Maki, 1974]. Not only does ethanol exert similar and additive CNS depression, but it also may facilitate the absorption of benzodiazepines. The implications of this are decreases in driving performance, alertness, coordination, and mentation [J*Forney & Huges, 1964; J*Linnoila & Hakkinen, 1974]. Alcohol increases the absorption rate of anxiolytic sedatives [J*Hayes et al., 1977; J*MacLeod et al., 1977], and they thus reach the CNS sooner and in higher concentrations than when taken without alcohol. The danger is compounded by the fact that toxic and lethal levels for such drugs are actually lower in the presence of alcohol, an expression of drug potentiation rather than simple summation. Alcohol and anxiolytic sedatives exhibit cross-tolerance. One practical implication of this is that anxiolytic sedatives can be substituted for alcohol without treating the chemical dependency problem. Another implication is that patients with the alcohol withdrawal syndrome can be effectively sedated with anxiolytic sedatives.

Alcohol & Sedative-Hypnotics

Ethanol, being an excellent solvent, increases the rate of absorption of sedative-hypnotic drugs, and thereby hastens the dissolution step, which is the rate-limiting step in drug absorption. Acute use of alcohol inhibits phenobarbital metabolism [J*Forney & Huges, 1964; J*Rubin et al., 1970]. Chronic use of alcohol increases the rate of metabolism of barbiturates through hepatic enzyme induction [J*Linnoila, Saario & Maki, 1974; J*Mould, Curry & Binns, 1972]. The simultaneous acute use of alcohol and sedative-hypnotics can cause an increase in CNS depression, sedation, incoordination, ataxia, and a feeling of drunkenness. In large doses, the combination can cause nystagmus, respiratory depression, coma, and death [J*Newman & Newman, 1956]. Cross-tolerance occurs between alcohol and other sedative-hypnotics. This means that sedative-hypnotics can be substituted for alcohol in patients physically dependent on alcohol.

Alcohol & Major Tranquilizers

When taken simultaneously with alcohol, major tranquilizers add to the CNS depression caused by alcohol. Orthostatic hypotension often occurs when alcohol and major tranquilizers are taken together. Cardiac arrhythmias are more common when patients combine major tranquilizers with alcohol than with either drug alone. Alcohol has been reported to cause akathisia and dystonia when taken in combination with phenothiazines [J*Lutz, 1976].

Alcohol & Marijuana

When taken acutely, marijuana potentiates the CNS depressant actions of alcohol. A patient showing signs of alcohol intoxication that do not correspond to the blood alcohol concentration (BAC) may have taken marijuana or other drugs, for example, sedative-hypnotics.

Alcohol & Tricyclic Antidepressants

When alcohol is consumed acutely with tricyclic antidepressants (TCADs), the sedation of the TCAD is potentiated [J*Landaver, 1969; J*Lockett, 1965; J*Seppala et al., 1973]. Chronic alcohol use leads to an increase in the rate of hepatic metabolism of the TCAD. TCADs delay gastric emptying and thus decrease the absorption rate of orally administered alcohol. Both TCADs and alcohol increase the incidence of cardiac arrhythmias. Orthostatic hypotension induced by TCADs is exacerbated by alcohol.

Enzymatic Blockers

The intermediary metabolism of alcohol provides a major source of drug interaction; several agents are known to interfere with this process. The most common site of interaction is the enzymatic degradation of acetaldehyde (ALDH), although there are drugs which inhibit the initial reaction catalyzed by ADH (e.g., pyrazole). ALDH activity is decreased by oral hypoglycemic agents (e.g., chlorpropamide, tolbutamide), as well as some antibiotics (e.g., metronidazole, chloramphenicol, quinacrine, isoniazid) see Table 1-7. The most pronounced effect of this kind, however, is exhibited by the drugs disulfiram (Antabuse [R]) and calcium carbimide (Temposil [R]). Both of these are employed for this very purpose in the treatment of alcoholism, though the latter is not available in the United States. A most disagreeable reaction occurs when alcohol is consumed by a patient taking disulfiram, due to accumulation of the toxic intermediate acetaldehyde. Cardiovascular manifestations are most profound, with a simultaneous rise in blood pressure and heart rate (noticeable palpitations), accompanied by vascular engorgement, flushing of the face and neck, pounding headaches, dizziness, nausea, vomiting, and extreme shortness of breath. It is essential that in the process of taking disulfiram, the recovering alcoholic express a sincere commitment to sobriety, in spite of the temptation to drink. Conversely, a waning sense of commitment is signaled by the patient's discontinuation of the drug well in advance of a drinking episode, for the patient knows full well that the long half-life of disulfiram makes it effective for 5-7 days after the last administration. With regard to drug interaction, it should be further noted that certain substances impair the effectiveness of disulfiram itself (e.g., high doses of vitamin C), and so the devious patient can "beat the system." Specific details about drug interactions with alcohol can be found in [J*Anonymous, Medical Letter, 1981b].

COMMENTS: Drug-Hypnosedative Interactions

Drug interactions with barbiturates occur frequently. Two essential mechanisms are involved in the process: potentiation of the depressant actions, and interference with drug metabolism via hepatic mixed-function oxidase enzymes. Alcohol, opiates, and other hypnosedatives may all be responsible for additive or even supra-additive central and respiratory depression. Drug metabolism is altered as a direct consequence of induced enzyme synthesis, that is, barbiturates stimulate synthesis of new metabolic enzymes. Effectiveness of other drugs can be markedly diminished as well, due to their accelerated rate of elimination (shortened half-life). Prominent among these are steroids, anticoagulants, anticonvulsants, and tricyclic antidepressants.

Chronic, excessive use of barbiturates and related sedatives (e.g., methaqualone, glutethimide, and methypylon) promotes the development of tolerance and dependence. It is not uncommon to find habituated users consuming 1.0-1.5 grams of pentobarbital or similar quantities of other drugs daily. When intoxicated, these

TABLE 1-7: Drugs which block Aldehyde Dehydrogenase^a

disulfiram (Antabuse)	citratated calcium carbimide (Temposil)
metronidasole ^b (Flagyl)	chloramphenicol ^b (Chloromycetin)
phentolamine ^b (Regitine)	griseofulvin ^b (Fulvicin)
tolbutamide ^b (Orinase)	chlorpropamide ^b (Diabinese)
furazolidine ^b (Furoxone)	quinacrine ^b (Atabrine)
nitroglycerine ^{b,c}	isosorbid dinitrate ^{b,c} (Isordil)

^a From [B*Becker, Roe & Scott, 1974, pp. 23-25]

^b These drugs produce only a mild reaction when one occurs.

^c From [J*Towell, et al., 1984]

people will be sluggish and dull in thought, speech, and body movement. They may appear morose or irritable with labile affect and, at times, frank paranoid ideation.

Persons on chronic sedatives as anticonvulsants must be wary of additive or potentiation effects of other drugs in the CNS depressant class. While the acute effects may be increased drowsiness and hazard while operating machinery, when the other depressant is removed, seizure threshold will be lowered.

COMMENTS: Drug-Marijuana Interactions

Because of the widespread use of marijuana use in our society today, drug interactions with the substance are most important. Two primary modes of interaction occur, namely potentiation of pharmacologic action, and interference with drug metabolism. Sedative drugs and others that depress CNS activity will be enhanced by coadministration of marijuana because it, too, has a sedative quality. A competitive interaction occurs with drugs that depend upon the hepatic mixed-function oxidase enzyme system (P⁴⁵⁰) for their metabolism. Therefore, the action of drugs, such as phenobarbital, desipramine, and others, may be further enhanced. It is a source of some solace that the margin of safety ("therapeutic ratio") is quite favorable for marijuana. An extremely large dose is necessary to produce frank morbidity or lethality, and this has never been reported.

Marijuana & Barbiturates

When marijuana is taken with barbiturates, there is an increase in subjective response to the barbiturates, with a corresponding decrease in motor performance [J*Dalton et al., 1975]. Marijuana increases the sleeping time induced by barbiturates [J*Sofia & Knobloch, 1973]. The interaction between marijuana and

barbiturates is time-dependent, in that stimulation occurs shortly after smoking marijuana. This is usually followed by sleepiness, drowsiness, and sedation. Additive depression of respiration may occur [J*Lockett & Milner, 1965]. Thus, in the early phase of marijuana intoxication, stimulation may occur that will slightly antagonize the effect of barbiturates, whereas later, the marijuana-induced sedation would interact synergistically with the sedation induced by the barbiturates.

Marijuana & Amphetamines

Marijuana and amphetamines, when taken together, synergistically increase heart rate and blood pressure [J*Evans et al., 1974]. It is reasonable that marijuana increases the stimulant activity of amphetamines because, in the early phase of marijuana intoxication, there is sympathetic stimulation and epinephrine release.

Marijuana & Anxiolytic Sedatives

Additive sedation results with the concomitant use of marijuana and the anxiolytic sedatives. In animals, marijuana potentiates the respiratory depression caused by benzodiazepines [B*Smith, 1976]. It is unlikely that this interaction is of clinical significance, except in massive overdoses of both drugs.

Marijuana & Other Drugs

Marijuana, when combined with physostigmine, causes an additive increase in heart rate [J*Kiplinger et al., 1971; J*Rosenblatt et al., 1972]. The interaction of THC and diazepam produces increased drowsiness and sleepiness, and a shift to the right of the carbon dioxide dose-response curve [B*Smith, 1976]. THC increases diazepam-induced sleeping time [J*Stone, McCoy & Forney, 1976]. Atropine interacts with marijuana to produce an unexpected pressor response, and tachycardia. Although the exact mechanism of this response is not known, the fact that it occurs in man is well established [J*Benowitz & Jones, 1975].

Marijuana & Narcotics

Although marijuana has been reported to increase narcotic-induced analgesia in animals [J*Boxbaum, Sanders-Bush & Efron, 1969], the clinical relevance of these observations remains to be elucidated. In animals, marijuana has been demonstrated to potentiate the respiratory depression induced by morphine [J*BeHuille, Swanson & Kamel, 1975; Johnstone et al., 1974]. There is probably little significance to this interaction.

COMMENTS : Other Drug Interactions**Pentazocine & Tripeleennamine**

The intravenous administration of a mixture of ground-up and partially dissolved pentazocine and tripeleennamine tablets is a drug-using practice that is common to most large cities in the United States [J*Butch et al., 1979]. The street mythology is that, when combined, these two drugs produce a heroin-like "high." This assertion is difficult to confirm or deny. The antihistaminic action of tripeleennamine may decrease some of the foreign body reaction and histamine release caused by the injection of pentazocine and particulate matter.

Benzodiazepines & Antihistamines

The antihistamines have sedative side effects which can be used in place of sedative drugs, and occasionally is used in sedative withdrawal. Cimetidine may potentiate the benzodiazepines [J*Cohen, 1983].

Opioids

The clinically significant drug interactions involving methadone are primarily of interest to the physician caring for methadone-maintained patients. However, such patients may present themselves to physicians and other health professionals in a wide variety of clinical settings, ranging from labor and delivery, to fractures. Phenytoin and rifampin increase the rate of metabolism of methadone, resulting in withdrawal [J*Finelli, 1974; J*Kreek et al., 1974]. Anticholinergics add to the constipation and urinary retention, and may cause adynamic ileus. They also antagonize the analgesic effects [J*Christensen & Gross, 1948; B*Goodman & Gilman, 1975; J*Roberts, 1968]. Neostigmine increases narcotic response [J*Christensen & Gross, 1948]. Monoamine oxidase (MAO) inhibitors have the capacity to potentiate both the depressive and analeptic effects of opioids [J*Anonymous, 1977; J*Evans-Prosser, 1968; J*Glover, 1967; J*Jacobson, 1965; J*Sargant, 1975]. Phenothiazines cause hypotension in patients on methadone [B*Goodman & Gilman, 1975], and may potentiate the depressive effect. CNS depressants potentiate the CNS depression of opioids, [J*Lasagna, 1965], while lithium enhances methadone effect.

Narcotic antagonists also precipitate withdrawal [J*Beaver et al., 1966; J*Bellville & Fleischli, 1968; J*Calimlim, 1973; J*Resnick et al., 1977; J*Tornabene, 1974]. One interaction of clinical significance is the consequence of combinations of opioid drugs where one of them is a mixed agonist/antagonist (ma/a) such as buprenorphine (not yet sold in US), butorphanol (Stadol), nalbuphine (Nubain), pentazocine (Talwin). Simultaneous administration of a ma/a with another agonist can block the other drug's agonist action [J*McGivney & Crooks, 1984]. In patients

who are physically dependent upon an opioid, administration of a m/a opioid can lead to sudden precipitation of an opioid abstinence syndrome [J*Fultz & Senay, 1975].

Drug Interactions

As stimulants, amphetamines interact with other drugs in a predictable fashion. When taken with other stimulants, they produce hyperarousal; when taken with certain antidepressants (monoamine oxidase inhibitors), cardiovascular instability and severe hypertension may ensue; and with depressants, a counteractive influence may be anticipated. Depressants are often used in a purposeful fashion by the stimulant abuser in order to terminate a "speed run." Combining the two physiologically opposite drugs at this point prevents the disagreeable and disconcerting "crash" that accompanies precipitous termination.

Care in prescription of certain drugs coincident with the use of cocaine is similar to that of amphetamines because of detrimental synergy (e.g., monoamine oxidase inhibitors). Furthermore drugs, which by themselves lower the seizure threshold, may assist cocaine in realizing its convulsive potential.

Sedatives & Stimulants

Although some antagonism occurs at low doses, at higher doses the depressant effect of the barbiturate predominates. Caffeine may antagonize or reverse the anxiolytic affect of the benzodiazepines, and should be ruled out before treating with a benzodiazepine [J*Cohen, 1983].

Tobacco

Cigarette smoking increases the rate of hepatic metabolism for a number of drugs, including chlordiazepoxide, diazepam (and presumably other benzodiazepines metabolized by the liver), propoxyphene, pentazocine, and chlorpromazine [J*Jick, 1974; J*Keeri-Szanto & Pomeroy, 1971]. Hence, cigarette smoking may decrease the potency of benzodiazepines [J*Cohen, 1983].

CONCLUSION

A knowledge of the more common drug interactions occurring between drugs of abuse can help eliminate unexpected reactions to drugs administered therapeutically. Obtaining a complete and accurate drug history is essential to preventing the occurrence of adverse drug interactions in substance-abusing patients.

CHAPTER 2: THE PATHOPHYSIOLOGY OF ETHANOL ABUSE

By James J. Lukes & Michael R. Liepman

OVERALL OBJECTIVES

The resident should be aware of the pathophysiology of various common conditions which are secondary to ethanol abuse so as to be able to understand how alcohol ingestion harms one's physical health, to counsel patients and their families about health consequences and risks associated with drinking, and to educate the community.

THE LIVER

LEARNING OBJECTIVE: Hepatic Pathophysiology

The learner should understand the progression of alcoholic liver disease and the relationship between ethanol use and liver pathophysiology. The key to early intervention and prevention of further damage is to be able to recognize early signs of liver damage and to sound the alarm effectively with patient and family.

COMMENTS: Hepatic Pathophysiology

There is a close correlation between per capita ethyl alcohol consumption and cirrhosis mortality [B*Fisher & Rankin, 1977; B*Rankin et al., 1975]. The vulnerability to liver disease may vary among individuals, but among chronic heavy drinkers, cirrhosis risk seems to be directly related to the lifelong volume of ethanol consumed. Although liver disease is not the commonest cause of mortality and morbidity, it is a preventable consequence of chronic excessive drinking. When patients show early evidence of liver damage, the family physician should be able to explain its meaning to the patient and family in the context of risk of future, continuing liver deterioration. Once the liver disease has progressed, the physician will need to be able to help the patient and family adjust to the disability that cirrhosis produces so as to maximize the patient's functional capacity and comfort, and to minimize the need for hospitalization [B*Gitnick, 1979].

Alcoholic liver disease is usually classified histologically into one of three categories [B*Scheuer, 1960]:

1. hepatic steatosis or fatty liver;
2. early fibrosis or alcoholic hepatitis; and
3. Laennec's or alcoholic cirrhosis.

Contrary to the mythology in the lay community, nutritional deficiency is not a prerequisite for the development of alcoholic fatty liver [J*Korsten & Lieber, 1979]. Ethanol is metabolized in the hepatic mitochondria, and nicotinamide adenine dinucleotide (NADH) is generated [B*Lieber & DiCarli, 1977]. The excess hydrogen ion (increased NADH/NAD⁺ ratio) thus formed stimulates fatty acid synthesis. The ethanol replaces the fatty acids as the energy source, and thus fatty acid oxidation decreases. This results in the deposition of fatty triglycerides in the liver; these cause hepatomegaly and liver tenderness [J*Lieber, 1978; J*Mendelson & Mello, 1973]. Biopsy of the liver at this stage would show fatty vacuoles within the liver cells. Uncomplicated fatty liver disease rapidly resolves itself with abstinence.

With repeated toxic insults to the liver parenchyma, the cellular contents are released into the bloodstream, including the liver enzymes, such as serum glutamic-oxaloacetic transaminase (SGOT), serum glutamic-pyruvic transaminase (SGPT), lactic dehydrogenase (LDH), and serum gamma-glutamyl transpeptidase (SGGT) [J*Casey et al, 1975; J*Konttinen, Martel, & Louhija, 1970]. In screening for acute alcohol-induced damage, SGOT is the most sensitive enzyme assay because it is released during heavy drinking both from muscle and liver cells. However in screening patients who may have stopped drinking several days ago, SGGT is superior because its level rises so high above baseline (up to twenty-fold) and its return to baseline may take two or three weeks after a bout of heavy drinking [J*Goldberg & Martin, 1975; J*Lamy et al., 1974 & 1975; J*Rosalki & Rau, 1972; J*Zein & Discombe, 1970]. In contrast, the other liver enzymes will reflect only more severe liver damage and for a more limited time after cessation of drinking. For the family physician SGGT can be a handy tool to identify concealed heavy drinkers in the practice and to demonstrate to these patients and their families that the alcohol is damaging their bodies. The body responds to this damage with an inflammatory reaction. This is probably when the fibrotic process begins. If there is a fairly large amount of damage, there may be temporary disruption of liver function, producing clinically recognizable signs such as jaundice, hypoalbuminemia, coagulopathy (based on disrupted vitamin K metabolism), and bile deficiency. Biopsy of the liver at this stage will show inflammation, fibrosis and damaged cells. An episode of alcoholic hepatitis generally resolves itself over time with abstinence and good nutrition. The liver may sustain a permanently greater sensitivity to future toxic or infectious insults.

Chronic, repeated insults to the liver, whether toxic or otherwise, seem to lead to accumulation of scarred (fibrotic) regions. The accumulation of the scars seems to affect adversely the flow of fluids (i.e., bile, blood, lymph) as they pass through the liver. Because most blood from the gut normally passes through the liver via the portal vein before entering the systemic circulation, gastrointestinal venous blood backs up, increasing portal venous pressure. This causes distention of gastrointestinal collateral vessels (esophageal varices and hemorrhoidal veins), and may cause splenic enlargement and hypersplenism. The hepatorenal syndrome may also be related to this,

although its mechanism remains a mystery. Trauma to the distended collaterals may lead to excessive and difficult-to-control hemorrhage, especially when a coagulopathy coexists. These veins are difficult to tamponade, because of their location. They also bleed at much higher pressure than typical veins. The decreased flow of fluids through the liver is also associated with reduced liver function, perhaps because of decreased delivery of nutrients and removal of toxins. Hence, it is characteristic to find the following liver functions disturbed in cirrhosis.

1. Decreased production of serum albumin leads to peripheral edema, ascites, and alteration of the potency and distribution-volume of albumin-bound drugs. The patient may lose the ambulatory function and skin may break down. In this context, salt and fluid loads are poorly managed by the kidneys. Hypertension may be more difficult to manage.
2. Multiple drug regimes for managing illness may become very unstable with minor changes in albumin level or changes in dosage of any one of the drugs, through alterations in the free:bound ratios by competitive binding to albumin or slight changes in catabolic rates.
3. Leakage of serum proteins into the abdominal cavity produces a mechanical obstruction to blood return via the inferior vena cava, to lymph flow from the lower extremities, and to normal diaphragmatic excursion (especially problematic in emphysematous patients). The accumulation of abdominal ascites may provide a physiological "third space" where drugs may accumulate over an extended time interval, producing a situation where drug levels may be slower to rise and fall with changes in dosage.
4. Disrupted vitamin K metabolism leads to coagulopathy and prolongation of the prothrombin time test. Excessive bleeding may result.
5. Disrupted amino acid and urea metabolism interferes with normal body maintenance and repair, as well as with the removal of toxic waste ammonia as urea. Gastrointestinal absorption of protein loads (as in high-protein foods or blood from upper GI bleeding) may lead to hepatic encephalopathy.
6. Disrupted glucose metabolism and glycogen storage may contribute to episodes of hypoglycemia [J*McMonagle & Felig, 1975; J*Phillips & Safran, 1971].
7. Decreased toxic catabolism may lead to higher levels and slower elimination of drugs and bilirubin, which require glucuronide conversion. Alcohol catabolism may also slow. These changes mimic reduction of tolerance to alcohol and other drugs.

8. Increased diversion of gastrointestinal blood through non-portal vein collaterals permits oral drugs to be distributed into the systemic circulation more rapidly and in higher concentrations without passing through the liver. This necessarily alters drug (including alcohol) potency.

If these conditions are superimposed upon the presence or absence of coexisting alcoholic hepatitis, the clinical condition of the patient will appear to wax and wane as the drinking starts and stops, and as the person recuperates. It is clinically quite tricky to deal with such a changing host, especially when several medications are being prescribed simultaneously.

TEACHING HINTS: Hepatic Pathophysiology

In a seminar setting, residents can be taught about liver pathophysiology by having them read articles [B*Fisher & Rankin, 1977; B*Gitnick, 1979; B*Korsten & Lieber, 1982; J*Lieber, 1978; B*Lieber & DiCarli, 1977] and present them to each other. The residents might also be encouraged to examine histology slides that are available [A*Ciba or A*Bonkowsky, 1981] and the accompanying booklet on liver diseases to determine whether these might be useful in explaining liver disease to patients and family members as a part of patient education.

In a clinic or hospital setting, when a resident takes responsibility for treating a patient with alcoholic liver disease, he or she might be prompted to explain to the patient and family how the pathophysiology of the liver disease manifests itself symptomatically. The resident could also be asked to explain to the preceptor or colleagues how liver disease affects the patient and how it should influence clinical decisions. The teacher or other team members could correct any errors, or the resident could be directed to read up on any areas of misunderstanding. If the resident encounters a patient with liver disease of unknown etiology, the preceptor may wish to observe whether the resident asks about drinking.

In the clinic, if the resident has examined a patient for routine health maintenance, the preceptor might inquire about whether a negative alcohol history and a normal liver were found. In this case, it might be helpful to debate which enzyme study is most sensitive in revealing subtle liver damage. If one is evaluating the overall health status of a patient in routine health maintenance, one might debate with the resident how far one should go to evaluate drinking patterns and their health effects in order to prevent cirrhosis and other alcoholic liver diseases?

While on a hospital rotation, the resident might survey the patients with liver disease to determine the proportion who suffer from alcohol-related liver disease. Likewise, the resident might review the chart of all inpatients on the service to determine the prevalence of abnormalities associated with liver dis-

ease. The resident could be asked to compare the utility of SGOT versus other liver enzymes as a screening tool in hospitalized patients [J*Casey et al., 1975; J*Combes et al., 1977; J*Goldberg & Martin, 1975; J*Konttinen, Hartel & Louhija, 1970; J*Rosalki & Rau, 1972; J*Zein & Discombe, 1970]. Once patients are identified as having liver disease, the role of alcohol should be considered in their diagnostic evaluation.

The resident might be asked to recommend the best method to determine the extent of liver damage (e.g., liver-spleen scan, CT scan, abdominal ultrasound, liver biopsy, BSP retention, etc.), so as to be able to realistically appraise the patient of this and warn patient and family of the future prognosis if drinking continues or stops. One might role play with the resident a patient and/or family wanting to know what to expect in the future. One should discourage the use of scare tactics that do not border on realism (e.g., "If you ever take another drink, you will surely die instantly."). On the other hand, one should also confront residents who talk only about the alcohol in terms of the liver. For example, the resident who says: "You should really cut down on your drinking" is correctly advising the patient about how to protect one's liver, but if the patient has alcoholism, cutting down may mean reducing intake by 20% which would provide little benefit, or it may be seen as an unempathic or unrealistic expectation by the family who realize that it is impossible for the alcoholic to significantly and reliably cut down without totally abstaining.

It is also important for the resident to understand the pharmacokinetic implications of liver disease in its various stages. For example, if a patient has early subclinical cirrhosis (i.e., liver appeared coarse on abdominal ultrasound but there is no ascites, no hyperbilirubinemia, and only a transient elevation of liver enzymes was observed at admission), what will happen to his phenytoin levels if he starts and stops taking disulfiram at will? What if he has clinical cirrhosis? What if he starts to drink heavily for a few days? It may not be possible for the resident or even the faculty to accurately predict the results in these cases, but it is important that the resident be able to think about the implications of pharmacotherapy in the real world with patients who have compromised liver capacity and who may intermittently add acute damage and transient dysfunction to underlying permanent liver damage.

It is important that the resident be aware of the anatomy of the liver and its blood supplies and the clinical implications of these. For example, the resident may be asked:

QUESTION 1: What does the presence of hemorrhoids indicate about the presence of esophageal varices?

ANSWER: It suggests that there was once enough portal hypertension to cause venous collateral dilation.

QUESTION 2: Why does tolerance decrease in drinkers with severe liver disease?

ANSWER: The venous collateral dilation permits newly absorbed alcohol and other drugs to enter the systemic circulation without first passing through the liver.

EVALUATION: Hepatic Pathophysiology

During a clinico-pathologic case (CPC) discussion or during clinical supervision following a patient visit, the preceptor can listen for evidence that the resident comprehends the spectrum of progressive stages of alcoholic liver disease. In routine health maintenance, does the resident demonstrate concern about drinking as a health hazard? Does the resident use liver health screening tests effectively? Does the resident give a patient a "clean bill of health" without screening for incipient liver disease or risky drinking patterns? In caring for patients with undiagnosed liver disease, does the resident consider alcohol as a potential contributing factor? In treating a patient with alcoholic liver disease, does the resident realistically consider the limitations of the alcoholic patient in devising the treatment plan? Does the resident counsel patients with early alcohol-induced liver damage to correct their underlying behavioral disorder properly? How well does the resident communicate knowledge of liver pathophysiology to the patient and family?

THE PANCREAS

LEARNING OBJECTIVE: Pancreatic Knowledge

The resident should understand the pathogenesis of acute and chronic alcoholic pancreatitis and the implications this has on its clinical management. The resident should know the connections between alcohol ingestion and diabetes mellitus.

COMMENTS: Pancreatic Knowledge

A patient seen during an attack of pancreatitis could have an acute, recurrent, or chronically relapsing disease. Because alcoholism is chronic, alcohol-related pancreatitis is usually of the chronic variety. Risk factors include malnutrition and the spree drinking pattern superimposed on years of chronic alcoholism. Constitutional factors, such as familial hyperlipidemia, presence of HLA antigen B-40, and anomalies of the pancreatic duct and the sphincter of Oddi, also play a role in risk [B*Korsten & Lieber, 1982].

The clinical features of alcoholic pancreatitis include recurrent attacks of emesis with abdominal pain, of increasing frequency but decreasing severity over time. The complications of

chronic pancreatitis include diabetes and malabsorption both of which can lead to a weakened and emaciated state. In diagnosing this chronic condition, a history of alcohol intake prior to attacks can be obtained serum amylase levels are usually in excess of 1100 IU/l [B*Korsten & Lieber, 1982].

Abstinence from alcohol becomes the most important determinant of prognosis, although attacks may recur even after several weeks of abstinence. While mortality only occurs in about 10% of attacks, the relapsing nature causes approximately 40-75% of victims who continue to drink to die within 10 years; in contrast, of those who quit drinking, only 20% die during this period. Predictors of mortality include age above 55, blood glucose above 200 mg/dl, leukocytosis over 16K, serum calcium below 8 mg/dl, and signs of hemoconcentration [B*Korsten & Lieber, 1982; J*Ranson et al., 1974]. The acute pancreatic attack usually resolves itself in time with supportive measures, such as intravenous fluids, proper nutrition and analgesics. Pancreatic pseudocyst and chronic, unremitting pancreatitis may require surgical subtotal pancreatectomy. After the acute attack has resolved, the patient's nutritional condition should be assessed. Pancreatic exocrine insufficiency can be treated with pancreatic extract, and fat-soluble vitamin deficits should be corrected. Permanent endocrine malfunction may require standard diabetic treatment with insulin administration.

Pathophysiology

It has been suggested that high concentrations of tissue ethanol cause the proteinaceous exocrine outflow of the pancreas to precipitate, temporarily obstructing pancreatic ducts [J*Sarles, 1974]. These plugs often contain protein, eosinophils, and they may become calcified [J*Sarles et al., 1965]. By this model, alcohol induces an increase in exocrine flow [B*Korsten & Lieber, 1982], which coupled with the plugged ducts, causes a build-up of intraductile pressure, which cause extravasation of exocrine digestive fluids into the pancreatic parenchyma, which could digest the tissue and stimulate an inflammatory response.¹ This would explain the excruciating pain and the digestive malfunction. The sequelae of recurrent attacks may include a pancreatic pseudocyst and loss of sufficient parenchyma to limit digestion and insulin production.

¹ Other models less well supported by scientific evidence suggest that (1) alcohol induced spasm of the sphincter of Oddi and conversely that (2) alcohol relaxes the sphincter of Oddi permitting duodenal or biliary reflux into the pancreatic ducts with resultant damage and scarring of the ducts [B*Korsten & Lieber, 1982].

TEACHING HINTS: Pancreatic Knowledge

In a seminar setting, the residents may be asked to read about alcoholic pancreatitis, its differential diagnosis, its clinical course, and its management [A*Bonkowsky, & Anderson, 1981; B*Howatt & Sarles, 1979; B*Korsten & Lieber, 1982]. They could each read different articles and then report to each other.

In a clinical setting, when a case of pancreatitis is being considered, the resident could be quizzed about this illness by a preceptor, asked to present the case at grand rounds or at a case conference, or be observed explaining the disease to the patient and family.

EVALUATION: Pancreatic Knowledge

During clinical teaching, the preceptor can watch the resident to determine the extent to which an understanding of pancreatic pathophysiology is reflected in clinical behavior. Does the resident consider the influence of chronic alcoholism on the pancreas? Does the resident properly explain the impact of continued drinking on the pancreatitis? Does the resident warn the patient and family about the risk of developing life-long diabetes mellitus and digestive disability or of dying from the illness?

THE CARDIOVASCULAR SYSTEM

LEARNING OBJECTIVE: Cardiovascular Toxicity

The learner should understand the effects of ethanol on the heart and cardiovascular system at both the cellular and clinical levels.

COMMENTS: Cardiovascular Toxicity

Ethanol interferes with mitochondrial respiration and alters the membranes of striated and smooth muscle. This inhibits cellular calcium uptake and binding by myocardial cells, which in turn depresses conduction and contractility. Ethanol inhibits or depresses both spontaneous and potassium-induced muscle contractions. Chronic ethanol consumption also results in a decline in the myocardial extraction of fatty acids from serum and in an associated increase in the extraction of triglycerides.

Ethanol contributes to the development of cardiomyopathy in which alcoholic patients, in the absence of coronary artery disease, develop insidious or acute onset congestive heart failure with diminished myocardial contractility. Alcoholic cardiomyopathy may be reversible if the patient abstains from drinking alcoholic beverages soon enough to avoid permanent cardiac dis-

tention. It is unclear why some patients manifest more prominent cardiotoxicity while others first sustain hepatotoxicity.

The clinical features of early alcoholic cardiotoxicity are subtle and easily overlooked. They may occur with isolated episodes of atrial fibrillation, nonspecific fatigue, or vague chest discomfort. As the severity of the cardiotoxicity progresses to cardiomyopathy, left heart failure will be manifested by exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea; right heart failure will be marked by venous distention and peripheral edema. Creatine phosphokinase (CPK) elevation may be seen. Low voltage changes are usually present on the EKG.

Another alcohol-related cardiac syndrome has been dubbed the "Holiday Heart Syndrome" because of its high prevalence after celebrations by binge drinkers [J*Ettinger et al., 1976; J*Regan, 1975; J*Regan et al., 1977]. This illness is usually reversible with abstinence or reduction in ingestion of alcohol. It usually occurs in the absence of coronary disease or cardiomyopathy. Atrial, ventricular or conduction abnormalities disappear within a day or two after the binge stops.

Ethanol acutely relaxes vascular smooth muscle. It has been prescribed in the past for angina pectoris. However, recent evidence suggests that the beneficial effects of coronary vasodilation may be a mixed blessing, because the drop in systemic blood pressure may cause tachycardia, increasing cardiac work. In untreated hypertensives, it may help by normalizing blood pressure and decreasing afterload. Some patients with coronary disease or arrhythmias are at high risk for myocardial infarction or arrest during the hyperadrenergic state associated with the alcohol withdrawal syndrome. In addition, nitrates react with alcohol in a manner similar to disulfiram (see Chapter 1).

The controversy over the influence of ethanol on the progression of atherosclerosis is still unsettled. Epidemiologic studies linking heavy drinking with hypertension, hyperlipidemia, myocardial infarction, and stroke are counter-balanced by studies finding no relationship between cardiovascular risk and drinking once the impact of tobacco smoking has been removed [J*Dyer et al., 1980; J*Haskell et al., 1984; B*Kannel & Gordon, 1970; J*Klatsky, Friedman & Siegelau, 1974 & 1979; B*Knott & Beard, 1982]. Studies suggesting a higher coronary risk in abstainers compared to moderate drinkers have been criticized for lumping together two groups² whose coronary risk factors other than alcohol intake may not be comparable to the moderate drinkers [J*Lieber, 1984]. Until recently, it was believed that studies showing an effect of ethanol on high density lipoproteins (HDL) suggested a mechanism for the alleged benefit of moderate drinking [J*Devenyi, Robinson & Roncari, 1980; J*Hartung et al.,

² those who abstain for health reasons (such as diabetics and recovering alcoholics) with those who abstain for religious or social reasons

1983]. However, recently Haskell and associates [J*1984] showed that the HDL₂ subfraction, which is protective of coronary vessels, is not² altered by moderate drinking; rather the HDL₃ subfraction, which has no relation to coronary risk, rises and³ falls with increases and decreases in drinking. Hence, a new mechanism will be needed to explain the epidemiologic findings.

Arterial hypertension is very common. Ethanol influences blood pressure in several ways. When the blood alcohol level (BAL) is rising, peripheral vasodilation and diuresis both produce a hypotensive effect. But during the postdrinking interval when the BAL is falling, sodium and fluid retention raise the diastolic blood pressure. There may also be an increase of adrenergic "tone" during the withdrawal period raising systolic blood pressure. It has never been shown whether this type of hypertension (i.e., recurring transient alcohol withdrawal-induced hypertension) contributes to cardiovascular (i.e., coronary artery disease, hypertensive heart disease) risk, but it definitely masquerades as idiopathic or essential hypertension in the office. The failure to recognize alcohol associated hypertension occurs most often when the patient is too embarrassed about drinking to wear alcohol on the breath during the examination or to inform the doctor of the extent to which drinking dominates one's life (i.e., the patient sobers up before coming to the office, even if the appointment must be cancelled and rescheduled several times).

TEACHING HINTS: Cardiovascular Toxicity

In a seminar setting, the residents might each read one article and then report back to the group. They might discuss the relevance of obtaining alcohol ingestion histories on patients with cardiovascular problems. They might be encouraged to estimate the probability that alcohol contributes etiologically to a cardiovascular patient's problems, given what is known about the epidemiology of drinking and the cardiovascular problem in question. A debate about whether hypertension or cardiovascular risk is related to alcohol use or abuse could stimulate careful thought about the issues which interact in such epidemiologic studies.

A debate about whether alcohol use should occur in the intensive coronary care unit could highlight the physiological consequences of drinking on the cardiovascular system and the psychological consequences of feeling anxious and deprived of one's usual daily drink.

In the office or hospital, a case of cardiac arrhythmia might be used to stimulate discussion of the differential diagnosis of rhythm disturbances. If the resident failed to obtain an alcohol ingestion history as part of the data base, the work-up should be judged incomplete. Similarly, in discussing a case of congestive heart failure, the possibility of alcoholic cardiomyopathy should be discussed. In a case of angina pectoris, the inquiry should include the impact of alcohol ingestion on the

chest pain. In an evaluation of hypertension, the role of alcohol should be presented as part of the standard data base. These are relatively common conditions, so there ought to be ample opportunity to discuss repeatedly the role of alcohol, such that when alcohol is an etiologic contributor, it will be rarely overlooked.

EVALUATION: Cardiovascular Toxicity

The following questions can be addressed during a case presentation that follows a patient visit or a fabricated CPC. Does the resident routinely obtain alcohol histories on patients with cardiovascular disease? Does the resident understand the impact of ethanol consumption at various doses on blood pressure, myocardial conduction and contractility, well enough to counsel a cardiac patient about wise use of alcohol? Does the resident utilize the alcohol history in assessing the cardiovascular condition? Does the resident consider the impact of untreated active alcoholism upon cardiovascular illness and its treatment?

THE SKELETAL MUSCLES

LEARNING OBJECTIVE: Skeletal Myopathy

The learner should be able to clinically recognize and understand alcoholic myopathy.

COMMENTS: Skeletal Myopathy

About 40% of chronic alcoholics have subclinical myopathies with elevations of serum enzyme levels. The creatine phosphokinase (CPK), aldolase, glutamic oxaloacetic transaminase (GOT), and lactate dehydrogenase (LDH) activities are increased in chronic alcoholics; the CPK level remains continuously above normal. Chronic myopathy (due to drinking binges) triggers muscle pain and tenderness. The onset of acute myopathy is abrupt, with a sudden appearance of muscular aching, cramping, and pain. Release of potassium from the damaged muscles produces hyperkalemia. An unusually severe case of acute muscle necrosis can result in myoglobinuria, renal failure, and ultimately, death. The duration of weakness after cessation of alcohol intake appears to be proportional to the duration of heavy drinking. Both chronic and acute myopathies can be reversed by correcting electrolyte imbalances and by instituting a well-balanced diet, fortified with high dose vitamins [B*Ferguson & Knochel, 1982].

Crush injuries to the muscles often follow an extended stupor during which the patient does not move about. Severe injuries of this sort can cause a serious myoglobinuria which may result in renal failure. Excessive potassium release into the bloodstream may cause cardiac arrhythmias.

The mechanism of myopathy is still unclear [J*Carlsson et al., 1969]. Hypotheses include electrolyte imbalances including deficiencies of magnesium, phosphorus and potassium, abnormalities in sodium or calcium transport, and changes in the cellular and organelle membrane structure and function [B*Ferguson & Knochel, 1982]. While the malnutrition hypothesis has been largely unsupported by scientific evidence, an animal model for acute rhabdomyolysis induced by acute malnutrition in the context of chronic heavy ethanol intake suggests that it may play a role [J*Haller & Drochman, 1980].

TEACHING HINTS: Skeletal Myopathy

In a lecture or seminar setting, readings and slides on muscle pathology could be presented. Each resident could be encouraged to read one article, and then present their findings to one another. They should consider the difficulty in recognizing skeletal myopathy unless the index of suspicion is high, given the more common causes of aches and pains seen in the primary care setting (e.g., febrile myalgias, trauma, muscle strain). They might debate the pros and cons of doing enzyme levels on every afebrile patient with myalgias and weakness.

In a clinical setting such as the office, the differential diagnosis for myalgias with weakness should include alcoholic myopathy. The preceptor might engage the resident in discussion about when to obtain enzymes and when to rely upon history.

EVALUATION: Skeletal Myopathy

In a clinical setting, the resident may be watched for attention to the alcohol history as part of gathering the data base on afebrile patients who complain of myalgias. If the patient is diagnosed as having alcoholic myopathy, how well did the resident explain what is the matter to the patient and family?

THE HEMATOPOIETIC SYSTEM

LEARNING OBJECTIVE: Hematologic Toxicity

The resident should be familiar with and understand the hematologic complications of alcohol overuse, and be able to treat such abnormalities.

COMMENTS: Hematologic Toxicity

The hematologic consequences of alcoholism [J*Colman & Herbert, 1980; J*Herbert, 1980] can be divided into three major types:

1. the red blood cell problems such as anemia [J*Conrad & Barton, 1980],
2. the coagulation problems [J*Cowan, 1980], and
3. the immune system problems [J*Liu, 1960].

Anemia may result from under-production through suppression of the bone marrow, or from blood loss through hemorrhage or hemolysis. Forty percent of hospitalized alcoholic patients exhibit a megaloblastic anemia characteristic of folate deficiency [J*Eichner & Hillman, 1971]. Direct ethanol damage to membranes of developing hematopoietic cells in bone marrow may cause progressive vacuolation in erythroblasts. Ringed sideroblasts are often present in the marrow of alcoholic patients with liver disease and folate deficiency. Folate and vitamin B₁₂ deficiencies may be present in severely alcoholic patients, but these are less common in well-nourished alcohol abusers. Megaloblastosis and macrocytosis may, however, be associated with normal serum, red cell, and liver folate concentrations. Well-nourished chronic alcoholics may have impaired intestinal folate absorption and decreased hepatic folate storage capacity coupled with an increased demand for folate. Ethanol also inhibits the conversion of absorbed folate to its active form, tetrahydrofolate, hence producing what appears to be folate deficiency yet folate levels may be within normal range and the clinical problem will not be correctable by folate supplementation unless the patient stops drinking [J*Lindenbaum, 1980; J*Sullivan & Herbert, 1964]. Alcohol also interferes with heme synthesis [J*Lumeng & Ting-Kai, 1974].

Chronic hemorrhagic blood loss from repeated trauma or gastrointestinal bleeding can deplete iron stores, producing an iron-deficiency anemia. In such cases, serum iron will be low and iron-binding capacity will be high.

Various hemolytic syndromes are associated with ethanol overuse. The erythrocytes of such patients have a mean corpuscular volume (MCV) greater than that found in blood from healthy controls without anemia. Macrocytosis occurs in the majority of chronic alcohol abusers and is associated with liver disease (although this may not be causal). Abstinence from alcohol is a prerequisite to replacing these large red blood cells with a new, smaller cell population. Membrane abnormalities secondary to changes in lipid content may also increase red cell fragility. The hemolysis associated with alcoholism occurs in the spleen when there is hypersplenism associated with liver cirrhosis [J*Cooper, 1980]. Hemochromatosis may result from the chronic hemolysis.

Alcoholics do not absorb vitamin K properly, and hence have a decrease in vitamin K dependent coagulation factors [J*Roberts & Cederbaum, 1972]. Liver disease may impair deactivation of clotting factors, which in turn may cause their rapid depletion [J*Fletcher et al., 1964]. Ethanol also impairs platelet synthesis, survival and aggregation [J*Cowan, 1980; J*Cowan & Graham, 1975]. Hence, the ultimate effect of such platelet changes is to cause prolongation of the bleeding time and to increase the risk

of hemorrhage. If hangover sufferers take aspirin, they may further interfere with platelet function and may contribute to acute alcohol-aspirin gastritis, a common bleeding syndrome in alcoholics. Other common bleeding sites include traumatized meninges, other injured body parts, and the gastrointestinal tract.

Alcoholics have compromised immune function, hence they are at high risk of becoming infected and they respond to infection poorly [J*Smith & Palmer, 1976; J*Liu, 1980]. Alcohol causes granulocytopenia via folate deficiency and direct bone marrow suppression of granulopoiesis, even when infection is present. In the presence of ethanol, granulocytes lose their normal chemotactic response used to locate the site of infection [J*Spagnuolo & MacGregor, 1975]. T-lymphocytopenia is also found [J*Bernstein et al., 1974]. This may be secondary to impairment of T-cell blastogenic transformation [J*Young et al., 1979].

Impairment of delayed hypersensitivity has been observed in cirrhotics [J*Straus et al., 1971]. In patients with liver disease, there are excessive levels of IgA, IgM, and IgE [J*Smith et al., 1980]. Suppressor T-cells apparently do not function to inhibit overly brisk responses to antigenic challenge. Fixed macrophages demonstrate impaired phagocytosis in the presence of alcohol [J*Liu, 1979]. Cirrhotic patients also have impaired IgM bacteriocidal activity [J*Fierer & Finley, 1979].

It is not unusual for an alcoholic patient to have several concurrent blood disorders. This may produce a confusing diagnostic picture. Unless the physician is prepared to entertain the thought that ethanol is the unifying factor behind the blood disorders, the pursuit of a diagnosis may prove fruitless. During a hospital stay, with little help from the physician, the patient may recover fully due both to abstinence from alcohol and eating a nutritious diet (containing folate, vitamin B₁₂, fat, methionine, calcium, and magnesium). Then, after the patient returns to the community and to drinking, the mysterious syndrome may recur.

TEACHING HINTS: Hematologic Toxicity

In a seminar setting, the residents could each read an article to present to one another [B*Chanarin, 1982]. Discussion could center around sorting out diagnostic entities when multiple blood disorders coexist. In the diagnostic evaluation of patients who may have an alcohol-related blood disorder, the residents could debate about methods of ruling out alcohol as an etiologic factor in ambulatory and inpatient settings. This might lead to some interesting discussion of cost-effectiveness and patient compliance. Or, consideration of a screening test for alcoholism might be entertained for inclusion in the standard battery of hematologic studies. The articles by Drum & Jankowski [B*1977 & 1982] suggesting the MCV as one of several screening indices for alcoholism might be discussed as they relate to the preventive medicine responsibility of the family physician.

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In a seminar, the residents could role play explaining the nutritional treatment of macrocytic anemia to a patient and spouse. Residents not participating directly in the roles could critique the manner in which the doctor brings alcohol into the discussion.

In a clinical setting, the preceptor may quiz residents on the alcohol consumption of their hematologic patients. One could ask the resident how long ago had the patient been drinking prior to obtaining the lab samples, in order to determine the impact of alcohol consumption on the test. When a hematologic diagnosis has been made, the preceptor may choose to evaluate how realistic the treatment plan seems when the patient's drinking patterns are considered.

In the laboratory setting, blood films and bone marrow biopsies from alcoholic patients may be used to graphically illustrate the impact of alcohol abuse upon cellular structure and function. Studying the slides in [A*Cornwell, 1981] can serve as a good review and update.

EVALUATION: Hematologic Toxicity

The preceptor can evaluate the resident's knowledge of alcohol induced hematologic toxicity by noting the nature of the diagnostic process, paying particular attention to consideration of the role of drinking in the etiology and treatment plan. Watching the resident explain this to the patient and family can be quite revealing. A case presentation of a complicated patient with overlapping blood disorders secondary to alcoholism can show how much the resident has learned.

THE CENTRAL NERVOUS SYSTEM

LEARNING OBJECTIVE: CNS Toxicity

The resident should be aware of and understand the acute and chronic effects of ethanol on the central nervous system and ethanol's association with brain damage and physical dependence.

COMMENTS: CNS Toxicity

Alcohol affects the central nervous system in many ways, some of which are desirable, others undesirable. Some effects are fully reversible, while others are permanently irreversible. Some effects alter mood and behavior, others affect consciousness and neurologic motor function. Some effects are noted acutely during initial intoxication, some after chronic intoxication, and still others are seen during withdrawal.

PATHOPHYSIOLOGY

In neurologic disorders associated with alcoholism, nutritional deficiency, trauma, infection, electrolyte imbalance, other neurotoxicity, intra-cranial pressure and vascular compromise all play mediating roles.

Neuropathological lesions associated with alcoholic brain disease are often unclear. The spectrum of lesions includes cortical shrinkage, periventricular lesions of Wernicke's encephalopathy and Korsakoff's psychosis secondary to thiamine deficiency, ventricular and subdural pressure syndromes following meningitis or subdural bleeding, cerebellar degeneration syndromes, strokes, brain tumors, brain abscesses and scars resulting from head trauma or other intracranial pathology [J*Calà & Mastaglia, 1981; B*McEvoy, 1982].

The Wernicke-Korsakoff Syndrome (W-KS) is caused by thiamine deficiency. Patients who develop the W-KS are usually of Mediterranean ethnic origin (they have a transketolase which binds less strongly to thiamine), and have been drinking alcohol without eating for several months [B*McEvoy, 1982]. Iatrogenic W-KS may be induced by acute administration of dextrose solution without thiamine added.³ The neuropathology suggests that there are atrophic lesions surrounding the ventricular system [B*McEvoy, 1982]. There is speculation that the cerebrospinal fluid acidity may play a role in the damage that is done. There is evidence that the affected nuclei involved in the memory dysfunction are primarily noradrenergic and that their normal function is to maintain attention.

CAT-scan studies have revealed radiological shrinkage of alcoholic brains (cerebral atrophy) which correlates with cognitive functional deficits of alcoholic dementia [B*Bergman et al., 1980]. Some of these abnormalities revert, at least in part, with several months of abstinence [J*Carlen & Wilkinson, 1980; B*Lishman, Ron & Acker, 1980]. Animal studies suggest that this reversible loss in brain mass and function is due to disconnection of the dendritic synaptic junctions rather than death of brain cells; hence the return of brain mass and cognitive function after cessation of drinking appears to be due to regeneration of dendrites with reconnection.

The toxic or withdrawal delirium of alcoholism has no characteristic anatomical lesions, but EEG changes and clinical signs help to make a diagnosis. Cerebrospinal fluid acidosis as-

³ Since chronic alcoholics are often thiamine depleted from dietary insufficiency or vitamin malabsorption, rapid intravenous infusion of dextrose solutions can be dangerous unless parenteral thiamine is simultaneously administered. Failure to do so can produce iatrogenic Wernicke's encephalopathy. Oral thiamine is not effectively absorbed until the GI tract has healed for several days.

sociated with the withdrawal delirium and EEG changes normalize over several weeks following abstinence [J*Carlen & Wilkinson, 1980]. Sleep disturbances mostly return to normal within a few weeks [B*Hartmann, 1982], but some EEG changes during sleep are persistent for as long as 21 months [J*Williams & Rundell, 1981].

One direct effect of drinking on the brain is the interaction of ethanol with lipid and protein components of the neuronal membrane, which acutely increases its fluidity. This action mimics the effects of other cerebral depressants, such as some general anesthetics. The fluidity change decreases ionic influx and efflux across the membrane, depressing neuronal conductance and neurotransmitter release [B*Blum, 1982]. However, chronic exposure is followed by alteration of the lipid composition of the neuronal membrane which reverses the former effect. This is believed to explain the acute toxic effects of alcohol as well as the development of tolerance and withdrawal.

Too rapid correction of dilutional hyponatremia in alcoholic patients has been blamed for central pontine myelinolysis. Although rare, this is a preventable severe brain disorder which has iatrogenic features.

When insulin dependent diabetic alcoholics derive their caloric needs entirely from alcohol, they may suffer severe hypoglycemia, which in turn may result in seizures and brain damage. Ethanol, like propranolol, may block the perception of hypoglycemia, hence interfering with the warning signs of impending insulin reactions.

Ethanol also interferes with biochemical conversion of neurotransmitters by competitively inhibiting the enzymes which convert alcohols to aldehydes and vice versa, hence altering the ratios of various neurotransmitters to their precursors and catabolites [B*Tewari & Carson, 1982]. One may reasonably speculate that such changes not only influence perception, mood and behavior, but that recurrent shifts in neurotransmitter ratios may stimulate cellular homeostatic regulatory responses to restore the original ratios. Indeed, studies have demonstrated alterations in receptor numbers and binding properties in association with alcoholism [B*Blum, 1982]. This has been suggested as one mechanism of tolerance and physiologic dependence. These changes may also account for personality changes, withdrawal reactions, and alterations in drug effects in alcoholics.

WITHDRAWAL SYNDROME

Sudden cessation of ethanol ingestion often induces a withdrawal reaction. Low serum magnesium levels have been found in some patients during withdrawal, allegedly secondary to the magnesium diuresis which occurs during drinking. Hypomagnesemia may increase neuroexcitability and predispose to convulsions during withdrawal. A respiratory alkalosis has been observed secondary to hyperpnea of unexplained origin in some alcoholics during withdrawal. Extremely low cerebrospinal pH has been observed in patients suffering from withdrawal delirium, although

the mechanisms and implications of this finding remain unclear [J*Carlen & Wilkinson, 1980]. Perhaps these latter two findings are related.

Although the majority of patients undergoing ethanol withdrawal are unlikely to suffer severely [J*Whitfield et al., 1978], the condition is potentially serious, even fatal, especially in the stressed host. The death rate from untreated withdrawal is small but significant, and usually occurs in patients who have infection, a fracture, or other severe concurrent illness. The measurement of withdrawal syndrome severity can be accomplished using one of the quantitative scales [B*Gross, Lewis & Hastey, 1974; J*Sellers & Kalant, 1976; B*Sellers & Kalant, 1982]. For a detailed discussion on withdrawal, see Chapter 5.

CHRONIC CNS CONSEQUENCES

Clinical neuro- and psychotoxicity of chronic alcohol ingestion includes:

1. headache syndromes [B*Saper & Magee, 1981],
2. cognitive and memory impairment, amnesia, and attention deficit disorders [J*Ryan & Eutters, 1980a],
3. coordination and gait disturbances,
4. toxic and withdrawal delirium [B*Sellers & Kalant, 1982],
5. withdrawal seizures,
6. insomnia and sleep disorders [B*Hartmann, 1982; B*Orr, Altshuler & Stahl, 1982; B*Rosett & Weiner, 1982; J*Williams & Rundell, 1981],
7. anxiety disorders,
8. hallucinations or misinterpretation of perceptions,
9. paranoid disorders such as the morbid jealousy syndrome or alcoholic paranoia,
10. delayed or arrested grief reactions (including post-traumatic stress disorders),
11. affective disorders with a very high suicide rate [B*Goodwin, 1982],
12. violent behavior, including family violence [J*Liepmann, 1980b],
13. risk-taking behavior, and
14. apathy towards the feelings and concerns of others.

The headache syndromes are probably pharmacologically related to histamine release induced by ethanol (cluster or histamine headaches) or to anxiety (tension headaches). Entries 2-6 above are primarily neurologic in nature. Entries 4-8 above are related to withdrawal from alcohol. The anxiolytic effect of ethanol on the mind probably contributes to the last five entries, since anxiety normally limits risk-taking and violence. Failure to adjust to traumatic events may indicate the normative role that anxiety plays in maturation.

Memory Disturbances

Alcoholic toxic amnesias (also known by laypersons as "black-outs") is a reversible, episodic, retrograde amnesia, limited to a clearly defined time interval during drinking until the body alcohol is partly eliminated (e.g., the patient cannot recall beyond the fourth drink consumed on the previous evening, including driving home). During the "forgotten" episodes, the person may behave normally or intoxicated. The absence of emotionally laden actions during a "blackout" helps rule out psychological repression as an alternative explanation for the amnesia. A history of recurrent similar amnesic spells, associated with heavy drinking bouts, also lends credence to a diagnosis of alcoholic toxic amnesia. The recollection of the missing information during subsequent drinking or during an amytal interview may suggest psychological defensive repression or state dependent learning mechanisms. In alcoholic toxic amnesia, these memories are not retrievable [B*Birnbaum & Parker, 1977].

Repeated amnesias may leave sufficient gaps in one's experience, especially surrounding one's own behavior during heavy drinking, that one may begin to suspect others of "making up stories." Denial that the "forgotten memories" could be important and that the accounts of others could be accurate, may contribute to the alcoholic paranoid state. Family members may begin to doubt their own sanity (especially children) unless they are aware of the amnesic phenomenon. The impact of recurring amnesias on the family can be very demoralizing. Children in this environment may learn to become dishonest or manipulative or to distrust their own perceptions.

The irreversible disturbances in memory storage which occur in alcoholics may either be secondary to Korsakoff's dementia or to alcoholic dementia [B*Birnbaum & Parker, 1977; J*Brandt et al., 1983; J*Ryan & Butters, 1980a & b]. Wernicke's encephalopathy is characterized by global confusion (delirium), ocular abnormalities (i.e., nystagmus, abducens palsies, and palsies of conjugate gaze), and ataxia (of stance and gait). Approximately 20% of patients with Wernicke's encephalopathy die, usually of infection. The recovery of the delirium following thiamine supplementation is rapid (i.e., 1-2 weeks), but is complete in only 15% of patients. The others progress into Korsakoff's psychosis which is characterized by anterograde amnesia (perhaps through hyperdistractibility), a patchy retrograde amnesia over the several years prior to diagnosis, apathy, inertia, lack of insight, with sparing of consciousness, verbal capacity, reasoning capacity, and grooming skills [B*McEvoy, 1932]. Confabulation is seen only in cases complicated by frontal lobe disease [J*Benson, 1978; J*Stuss et al., 1978]. The recovery of the ocular abnormalities (complete in only 40%) occurs within hours to days of first parenteral thiamine administration. The recovery of the ataxia occurs (complete in only 40% of patients) in a period of 1-3 weeks. In milder forms, the intentional rehearsal of information to be remembered helps to improve cognitive function, especially if word-associations are selected for each item to be recalled [J*Binder & Schreiber, 1980]. Clonidine

administration has shown to be helpful in improving memory storage in Korsakoff's psychosis [J*McEntee & Mair, 1980; J*McEntee, Mair & Langlais, 1981]. Memory retrieval performance may improve with clues during testing.

In alcoholic dementia, long-term memory may disappear in a retrograde fashion, with loss of memories from before the onset of the disorder. In addition, there are associated visuospatial deficits and a loss of short-term memory capacity. The neuropsychological deficits and the brain atrophy mimics accelerated aging of the brain at roughly double the normal rate [J*Ryan & Butters, 1980b]. In fact, moderate drinkers appear to sustain milder memory and cognitive deficits [J*Parker & Noble, 1977]. Recovery of the short-term memory impairment of alcoholic dementia appears to occur with extended abstinence, but the long-term memory deficit seems to remain even after 7 years of continuous abstinence [J*Brandt et al., 1983].

Short-term memory disturbances are also caused by excessive anxiety which interferes with concentration. Affective disorders can also affect memory. And the ingestion of sedative-hypnotic agents, especially the benzodiazepines, can interfere with short-term memory.

Sleep

The effects of alcohol on sleep of non-alcoholic persons is to reduce the amount of rapid-eye-movement (REM: the stage when dreaming occurs) sleep early in the night and to augment it later in the night. In exchange, there is an increase in slow wave sleep early in the night. However, if alcohol is repeated nightly for five days, tolerance seems to develop for this effect on REM sleep. In some studies there is shortening of sleep latency [B*Hartmann, 1982].

In alcoholics, initial drinking after a sober period seems to have the same effects as in non-alcoholic persons, namely a reduction in sleep latency, increase in slow wave sleep early in the night, and reduction of REM sleep. But since most alcoholics have a great deal of tolerance, even early drinking usually involves much higher doses, hence more extreme effects. As the drinking continues, tolerance develops to these effects such that the REM and slow wave sleep returns to baseline, though there may be more shifting from stage to stage with frequent awakenings throughout the night. The alcoholic may be uncertain whether he is having nightmares or is awake and hallucinating. Then, if drinking ceases, there is insomnia followed by hallucination with excessive REM during the hallucinations (as though the patient is dreaming while awake). This is eventually followed by a period of heavy sleep. Some residual sleep disorder may be present for months after withdrawal [B*Gross & Hastey, 1976; B*Hartmann, 1982; B*Pokorny, 1978; B*Williams & Salamy, 1972]. The sleep of the newborn whose mother drank during the prenatal period is also disturbed [B*Rosett & Weiner, 1982].

Fetal Alcohol Effects

The Fetal Alcohol Syndrome (FAS) and the milder Probable Fetal Alcohol Effects (PFAE) include irreversible brain dysfunction that may be associated with anatomic anomalies of the brain. It has been well established in animal models and has been observed in humans that the brain of the FAS child is smaller and performs below that of unaffected individuals in a number of functions [J*Streissguth et al., 1980; J*Streissguth, Herman & Smith, 1978]. The exact mechanism of this is unknown, but hypotheses have included nutritional deficiencies due to alteration of active transport of vitamins and amino acids across membranes, alteration in cellular migration in the organizing fetal brain, acetaldehyde toxicity on fetal tissues, and intrauterine hypoxic episodes secondary to umbilical arteriospasm [J*Mukherjee & Hodgen, 1982]. Kenneth Lyons Jones has reported the appearance of speech disorders in the children of mothers who were treated for premature labor with ethanol drips.

TEACHING HINTS: CNS Toxicity

The students could read and report in a seminar on articles relating to some element of the CNS toxicity of alcohol (A*Bernat & Victor, 1981; B*Birnbaum & Parker, 1977; J*Carlsson et al., 1979; B*Hartmann, 1982; B*McEvoy, 1982]. In particular, they might debate the controversial views on the premature aging hypothesis of cognitive loss in alcoholics. They might also debate implications of a proposed policy to prohibit any use of alcohol by airline pilots, nuclear submarine commanders, legislators, pregnant mothers, and executives of major industries.

In a CPC, an insulin dependent patient arrives comatose in the emergency department having consumed a large quantity of alcohol. The residents should be able to present the differential diagnosis and treatment plan for such a patient including both short- and long-term strategies. Issues to be covered should include:

1. diagnosis of etiology for coma, including alcohol or mixed overdose, post-ictal state, hypoglycemia, intracerebral hemorrhage, meningitis, dilutional hyponatremia or other electrolyte imbalance;
2. treatment of immediate coma including support of vital functions, narcan, dextrose infusion, anticonvulsants, antibiotics, correction of electrolytes, etc.;
3. caveats in these treatments such as preventing Wernicke's from dextrose infusion, preventing central pontine myelinolysis during electrolyte correction, preventing recurrence of coma by prematurely permitting the patient to escape observation after glucose bolus or narcan injection;
4. long-term attempts to obtain alcoholism rehabilitation for the patient.

In their hospital or clinic practice, they could be quizzed by the preceptor about the neurological deficits that could befall their alcoholic patients, and whether alcoholism could be the cause of neurological disorders in any of their patients. When a patient is discovered to have alcohol-associated neurotoxicity, the resident should be encouraged to read about it.

EVALUATION: CNS Toxicity

The mention of alcohol history as part of a diagnostic evaluation of any neurologic patient is good evidence that the resident has incorporated the alcohol intake as a relevant factor in the development of neurologic disease. Picking up subtle neurologic signs of impairment in alcoholic patients would also reflect an awareness of these factors. Quizzing the resident on these facts and their relationship to clinical patient care is another way to evaluate recall and understanding, though quizzing will not guarantee that such an understanding will be used in clinical practice.

In the case of the patient with a neurologic disorder known to be secondary to alcohol, or the patient who risks neurologic damage by continuing to drink excessively, observing how the resident explains this to the patient and family may facilitate evaluation of the clinical understanding of CNS toxicity. Clinical management of the patient with alcohol associated toxicity of the nervous system will also provide an opportunity for the preceptor to evaluate the resident.

Observing how the resident evaluates a patient with a sleep disorder or memory problems will reveal the extent of understanding of the relationship between drinking and these problems.

In family counseling, recognizing the possible effects that drinking might have on the feelings and behavior of children would reflect the resident's competence in this area.

ENDOCRINOLOGY & BIOCHEMISTRY

LEARNING OBJECTIVE: Endocrine/Biochemical Toxicity

The resident should recognize and understand the effects of ethanol on the endocrine system and on intermediary metabolism. The resident should know how to treat these disorders.

COMMENTS: Endocrine/Biochemical Toxicity

Ethanol can interact with the endocrine system at multiple sites, including the hypothalamus, pituitary, or the target gland itself. The peripheral metabolism of hormones can be altered by ethanol's direct effect on cardiovascular and hepatic blood flow,

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enzyme/cofactor alterations, or receptor dysfunction. Ethanol indirectly effects endocrine function by causing vitamin and caloric deficiencies, and by causing a withdrawal state.

SEXUAL FUNCTION

Disturbances of sexual function are frequently alcohol-associated endocrine problems. In male chronic alcoholic patients, both hypogonadism (including reduced libido, testicular atrophy, oligospermia, or azospermia) and feminization (with gynecomastia) are common. In females suffering from alcoholism, masculinization (with lowering of the voice, hirsutism, amenorrhea, infertility, and reduced sex drive) occurs. Liver cirrhosis prevents preferential detoxification of end-organ produced hormones characteristic of the opposite sex (i.e., androgens in females and estrogens in males). Ethanol also directly interferes with testicular androgen production and spermatogenesis. These hormonal effects contribute to the etiology of impotence and infertility in males suffering from alcoholism [J*Eagon, Porter & Van Thiel, 1983; B*Fadel & Hadi, 1982; B*Karacan & Hanusa, 1982].

CARBOHYDRATE METABOLISM

When ethanol is present in large quantities, carbohydrate metabolism is turned down so as to catabolize ethanol at the maximum rate. This may be mediated through limitations in the electron receptor NAD/NADH⁺. One consequence of this metabolic alteration is impaired gluconeogenesis and glycogen storage. Ethanol is a potent source of energy (7.1 Kcal/g), hence suppressing appetite. Pancreatic damage may reduce the quantity of available insulin, needed to facilitate cellular entry of glucose. A disproportionate number of alcohol abusers have carbohydrate intolerance. In the insulin-dependent diabetic alcoholic, depleted glycogen stores or poor liver perfusion may decrease the body's capacity to respond by glycogenolysis when ethanol fuel is depleted. Alcohol abuse should be suspected in diabetics who develop hypoglycemic episodes, especially at night, or who have difficulty maintaining reasonable control of their diabetes. Some patients develop alcoholic ketoacidosis from chronic excessive drinking. Alcohol suppresses the growth hormone response to hypoglycemia [B*Stokes, 1982].

Ethanol catabolism often creates a build-up of lactate via anaerobic glycolysis, which in turn causes the retention of uric acid, producing hyperuricemia and gout [B*Lieber, 1982].

THYROID

During withdrawal, the T₃ and T₄ levels were elevated but returned to normal shortly thereafter. The status of the relationship between alcohol and thyroid function is unknown. Study results are quite variable and have shed little light on clinically relevant findings [B*Stokes, 1982].

CHAPTER 2: PATHOPHYSIOLOGY OF ETHANOL ABUSE

PROLACTIN

Prolactin levels are higher in alcoholic males with liver disease, and when the estrogens are elevated, gynecomastia results [B*Stokes, 1982].

VASOPRESSIN

The diuresis following heavy drinking has been linked to ethanol induced inhibition of vasopressin release. This occurs only in the absence of hyperosmolality [B*Stokes, 1982].

STEROID METABOLISM

Ethanol may alter the activity of enzymes involved in the metabolism of steroid hormones. Chronic alcoholics may then develop features resembling Cushing's syndrome; these are characterized by facial mooning, proximal muscle wasting, excessive bruising, hypertension, and osteoporosis with vertebral collapse. Biochemically, both types of patients (Cushing's and alcohol-induced pseudo-Cushing's) have elevated plasma cortisol levels and increased urinary steroid loss; dexamethasone fails to suppress cortisol secretion. This syndrome disappears within days to weeks of stopping use of ethanol, but returns if drinking resumes [B*Stokes, 1982]. Ethanol also stimulates the pituitary to produce corticotropin releasing hormone.

DRUG CATABOLISM

Ethanol acutely competes with the catabolic pathways of most other drugs at the microsomal enzyme oxidative system (MEOS). However, chronic ingestion of large quantities of ethanol induces the MEOS to increase its catabolic capacity. Hence, an acute dose of ethanol is likely to potentiate most other drugs, but a chronic ingestion pattern is likely to weaken most other drugs by hastening their inactivation by oxidative mechanisms. It is difficult to predict exactly which process (i.e., competitive inhibition versus enzyme induction) will predominate when ethanol is present in an alcoholic. However, it is safe to say that when an alcoholic patient is treated with other drugs, the stability of their blood levels on any particular fixed dosage regimen is endangered by the unpredictable shifts in their MEOS reserve.

TEACHING HINTS: Endocrine/Biochemical Toxicity

In a seminar setting, the residents could read and present to one another about the various toxic mechanisms mentioned above [A*Adler, 1982; A*Smith, 1981]. They could discuss real or fabricated CPCs about patients whose care was complicated by their drinking. Three such cases follow:

CASE 1: John is a 53 year old man who is taking coumadin for prevention of recurrent phlebitis which had caused two prior hospitalization for pulmonary emboli. He presents with multiple large bruises without a history of trauma. His prothrombin time was within the therapeutic level only two days prior to the hospitalization when he came to the office for his weekly venipuncture. On his regular dose, it returns to the target level within several days of hospitalization. He swears that he has not altered his dosage and his wife agrees, having checked his pill count and having watched him take his medication.

ANSWER: One should be sure to discuss the reasons for the failure to recall the trauma, perhaps because it occurred during alcohol amnesia. The transient elevation of coumadin potency may be explained by a drinking binge not mentioned in the case presentation.

CASE 2: Betty is a 16 year old juvenile onset diabetic who has been managed successfully for three years on 28-30 units of Lente insulin and a 1500 Cal. ADA diet. She presents to the hospital in ketoacidosis, but is eventually discharged on the same dosage of insulin. During the hospitalization, she denied altering her diet, reducing her insulin dosage, or changing her level of activity; and no infection was found. Within three weeks, she was again spilling glucose intermittently in her urine and had three insulin reactions. On repeat hospitalization, she again was controlled adequately on 28 units of Lente insulin.

ANSWER: Betty is an adolescent who may be drinking with her peers to gain their acceptance, an important sign of autonomy in a 16 year old. However, she may be drinking enough alcohol to block glycogen storage (needed to combat insulin reactions) and to upset her caloric intake. Since it is illegal to drink at her age, she may be unable to comfortably tell her physician about it.

CASE 3: Bill is a 21 year old college student who presents with allergic symptoms of rhinorrhea, itchy hives and bronchospasm. He also complains of sinus headaches. He smokes cigarettes by history. He has a positive past medical history for allergic symptoms.

ANSWER: Allergic persons are relatively sensitive to changes in the histamine levels in the body. The resident might obtain a thorough dietary history including sources of histamine (e.g., sauerkraut, beer) and histamine releasers (e.g., chocolate, ethanol, strawberries) other than allergens. While tobacco smoke can trigger sinus irritation, this could be exacerbated by a sudden increase in ambient histamine levels in the body. The bronchospasm could relate to a sudden increase in histamine levels, or it could result from sudden cessation of heavy marijuana smoking.

In a clinical setting, the preceptor must stay alert to relevant opportunities to teach about these toxic syndromes. In teaching about the proper diagnostic work-up of endocrine and metabolic disorders, it is wise to include an environmental lifestyle portion to the history to ensure adequate information will be obtained to properly understand the role, if any, that chemicals play. Patients often do not realize that over-the-counter drugs, recreational chemicals and work-related environmental toxins have any relevance to health and disease, so it is necessary for physicians to play an aggressive role in seeking this information.

EVALUATION: Endocrine/Biochemical Toxicity

The residents could be asked to recount the multiplicity of ways in which alcohol interferes with the metabolism and the endocrine system. The best way to evaluate this is by watching for a toxicological approach in clinical problem solving.

THE FETUS

LEARNING OBJECTIVE: Fetal & Neonatal Toxicity

The resident should be familiar with the effects of alcohol, tobacco, and other drugs on the developing fetus. It is important to recognize damage secondary to substance ingestion and, more crucially, to counsel the pregnant patient and her family to avoid chemical pollution of the womb.

COMMENTS: Fetal & Neonatal Toxicity

The rediscovery of the damaging effects of alcohol on the fetus in the early 1970's reiterated the painful lesson of thalidomide twenty years before. Now many chemicals are being found dangerous to the developing fetus and all are being considered suspect until proven otherwise. The initial controversy about the Fetal Alcohol Syndrome (FAS) probably resulted from the protected status that alcohol has in our culture, coupled with the variability and subtlety of expression that is characteristic of this teratogen. Although most conventional teratogens are known by the narrow specificity of their damage, alcohol can damage a variety of tissues at a variety of times during embryogenesis and fetal life. In addition, variations in maternal drinking times and dosages join with differences in fetal vulnerability to produce a spectrum of probabilities for each type of damage. The picture becomes even cloudier when one adds to this the variations in the exposures to other conditions, such as smoking, malnutrition, and use of other drugs [J*Rosett, 1980; J*Kaminski, Rumeau & Schwartz, 1978; J*Kruse, 1984; B*Rosett & Weiner, 1982; J*Streissguth et al., 1980].

The morphologic damage to the fetus, the permanent impairment of growth, and the mental retardation all may lead to life-long disability for the FAS damaged child. This may produce added stresses to an already sick family. In addition, the neonatal withdrawal syndrome produces an extremely fussy child, who is even more difficult to care for than the average child. This can interfere with parent-child bonding during the all-important first weeks of postnatal life, and can permanently damage the family psychological milieu [B*Antonucci, 1983; B*Stack, 1983]. This could pose a major and perhaps insurmountable problem for a mother who already had problems with self-esteem and expression of feelings. She may be tempted to blame herself and relapse into (or continue) using drugs or alcohol to cope with the new baby's apparent disinterest in the new parents. A precondition for future child abuse or neglect could be established in this situation. The natural course of this problem could be altered by careful infant detoxification and parental support during the early days of postnatal life.

Drug ingestion other than alcohol during pregnancy has also been implicated in teratological abnormalities [B*Ferguson, Lennox & Lettieri, 1974]. The details of these problems will not be reviewed in this monograph. However, it is safe to say that with the increasing research in this area, one needs to keep abreast of the literature in order to stay updated to current knowledge. The most sophisticated research these days looks at drug ingestion critically along with nutritional factors and interactions between multiple environmental toxins.

Another issue of great importance and even greater confusion is that of drugs in the nursing mother's breast milk. Some drugs pass into the milk in reduced or enhanced concentrations. The relationship of the overall quantity of the drug in the milk, divided by the size of the infant, makes it tricky to determine whether there is any reason for concern about any particular drug. Inhalants, such as tobacco or marijuana, may be smoked in close proximity to the infant, putting the infant at risk in another way (e.g., children of smokers have a three-fold greater risk of pneumonia than children of nonsmokers), whether the infant is bottle- or breast-fed.

The father's contribution to this environment can best be dealt with when the father is included in the neonatal care. There may also be need to involve other significant persons in the neonatal environmental assessment and neonatal care. Fortunately, the family physician is more interested in and better equipped to approach these family prevention issues than the obstetrician.

TEACHING HINTS: Fetal & Neonatal Toxicity

The residents could devote a seminar or two to the review of literature and slides on the fetal teratological syndromes that appear in association with the ingestion of psychoactive chemicals [A*Rosett; A*Crisis in the Unborn].

The residents could also obtain copies of local laws and regulations concerning child protection. They might invite a child protection agent to discover how the local authorities deal with protection of unborn children from the damaging effects of maternal indiscretion in chemical ingestion. They could struggle with the legal conflict posed by substance abuse confidentiality laws which seek to protect the mother's right to remain concealed versus the child's right to be protected [J*Anonymous, 1979].

In a clinical setting, the resident studying obstetrics could be asked to survey expectant mothers to determine the proportion among them who have exposed themselves and their fetuses to risk. On a pediatrics rotation, the residents could try to identify children whose mothers have ingested psychoactive chemicals, and who show developmental or mental disabilities, structural anomalies, or a failure to thrive. A debate among a group of residents could focus on the ethical issues raised by the chemically dependent mother who placed her fetus at risk by continuing to abuse (or use) psychoactive chemicals during her pregnancy. (Be careful that this does not degenerate into an overly simplistic and self-righteous tirade against a hedonistic woman. Set up the debate with advocates for both the child and the woman.) A discussion could focus on the best time during the recovery process to break the news to the family that the new infant has been damaged by the mother's prior substance abuse.

An excellent teaching slide program [A*Rosett] is available for the residents to review; some of the slides might be used to develop a model community or patient education program, which could be offered to school health classes, parent groups, and those receiving prenatal care. The residents could debate what constitutes a safe level of exposure (to each of the psychoactive pollutants) for the fetus or nursing infant. This would demand attention to the research literature and would force the residents to consider the real difficulties in applying research to real-life situations.

EVALUATION: Fetal & Neonatal Toxicity

Residents caring for prenatal and pediatric patients should pay attention to chemical exposure histories. If this information is not readily apparent in their records and case presentations, the faculty preceptor may inquire about why this is not being taken seriously. In cases with a negative exposure history, the preceptor may be wise to play a skeptical role in challenging the resident's thoroughness and interviewing skills; many patients conceal their substance use from physicians in order to obtain acceptance and approval and to avoid embarrassment. Watching for stereotyping of patients (e.g., she's not the type who would drink during pregnancy, so I didn't feel the need to pursue this line of inquiry) or sloppy interviewing (on videotape or live observation or subsequent reinterview by the preceptor) should help curb false negatives. In cases with a positive history of exposure, the therapeutic and preventive plans should reflect a thoughtful approach to this challenging problem. Specifically,

the plan should include efforts to protect the unborn child and to help the patient-mother and other family members to recover from the addictive disorder with dignity.

EPILOGUE

This chapter attempts to bring the family practice educator up to date on some of the pathophysiologic mechanisms through which alcohol damages the health of the individual (and the family). The scope of this chapter does not permit total coverage of all of the complications of alcoholism. But it is hoped that the chapter will stimulate the educator to delve more deeply into the relevant literature, and thereby facilitate the training of residents and others into optimal care and management of alcohol- and other drug-dependent patients. A more complete listing of alcohol associated illnesses may be found in [B*Liepman, 1983]. Further information on a multitude of medical consequences of alcohol and drug abuse may be found in general references [B*Kissin & Begleiter, 1971; B*Lieber, 1982; B*Pattison & Kaufman, 1982; A*Project Cork; B*Wartenberg & Liepman, 1984].

CHAPTER 3: FINDING SUBSTANCE ABUSERS

By Michael R. Liepman

OVERALL OBJECTIVE

The teacher should be able to teach the signs and symptoms of alcoholism and other drug dependencies in order to facilitate early clinical recognition of the addictive disorders. In order to do this efficiently, the resident must be taught to have an appropriate index of suspicion, hence awareness of the prevalence of such syndromes is important. The resident must be taught to distinguish alcoholism and other drug dependencies from other associated behavioral conditions, mental disorders, social problems, and primary medical illnesses which may be caused or exacerbated by the addictive behavior, may be concurrent but unrelated to the addiction, or may be underlying risk factors. The teacher should also be able to teach the resident to assess the degree of progression of the chemical dependency.

EPIDEMIOLOGY

LEARNING OBJECTIVE: Epidemiology

The family physician should be able to calculate the proportion of his or her patients and families who are probably afflicted by or at risk of developing chemical dependence.

COMMENTS: Epidemiology

To achieve reasonable success in early recognition and diagnosis of substance abuse disorders, family physicians need to be aware of the prevalence of the addictions in their own practices. Alcoholism has been estimated to afflict roughly 10% of the population, but the prevalence in one's own practice may vary for several reasons [B*Mulford, 1982]:

1. The ethnic background and social milieu surrounding the clinic increases or decreases the prevalence [B*Cahalan, Cisin & Crossley, 1969; B*Westermeyer, 1982].
2. The age, gender or socioeconomic structure of the population served is dissimilar from that of the total national sample from which the estimates have been derived [B*Barnes, 1982; B*Filstead, 1982; J*Funkhouser, 1977; B*Maletta, 1982; B*Mihsara & Kastenbaum, 1980; B*Rice, 1982; B*Sandmaier, 1981; B*Wilsnack, 1982].
3. The patients who come to the clinic are more ill than the general population, and those who come more often are more ill than those who come infrequently [J*Pell & D'Alonzo, 1968; J*Poikolainen, 1983].

4. The criteria used to diagnose the addictive illness are different from those used by epidemiologists [B*American Psychiatric Association, 1980; B*Cahalan, Cisin & Crossley, 1969; B*DuPont, Goldstein & O'Donnell, 1979; B*Jacobson, 1976; B*Jellinek, 1960; J*Keller, 1960; J*National Council on Alcoholism, 1972; J*Seixas et al., 1976; B*WHO, 1964].

The prevalence of alcoholism in primary care clinic populations in two studies [J*Breitenbucher, 1976; B*Liepman, 1983 & 1984] varied from 9% in a university town to 44% in an inner-city clinic. In both samples, there were more alcoholic males than females. In both samples, frequent clinic attenders were over-represented. In hospitalized populations, the prevalence ranges from 20% to 30% depending upon other factors mentioned above [J*Funkhouser, 1977; J*Quinn & Johnston, 1976; J*Senior & Sloan, 1981; J*Westermeyer, 1978]. Usually about half of adult psychiatric admissions are alcoholic [J*Liepman & Maiorana, 1979; J*McCourt, Williams & Schneider, 1971; J*McLellan, Druley & Carson, 1978; J*Moore, 1977], though not necessarily recognized as such, and about half of juvenile psychiatric admissions are children of alcoholics.

Prevalence [B*Schuckit, 1979] of sedative drug (other than alcohol) use by adults is 15% each year: (a) 90% of hospitalized med/surg patients receive them; (b) 10% of med/surg and 30% of psychiatric patients believe themselves to have had sedative psychological dependence at one time; (c) many sedative drug abusers obtain their drugs legally from physicians; (d) hypnosedative and minor tranquilizer drugs constitute the largest pharmaceutical market in the US (10 billion doses/year in 1970); (e) many regular users of these drugs do not realize that they are addicted to them until they stop using them [B*Ray, 1978]. Bromides, chloral hydrate, meprobamate, methaqualone, barbiturates, and now the benzodiazepines each, in turn, have been introduced as the non-addicting sedative, have gone through heavy commercial promotion, then during periods of wide usage have been shown to be addicting and harmful if abused, and ultimately have earned greater restrictions on production or distribution [B*Ray, 1978].

Stimulant drugs are found in the urine of 15% of all psychiatric hospital admissions [B*Schuckit, 1979]. The specific drugs used have varied with changes in availability and fashion from amphetamines in the 1960's to cocaine and phenylpropanolamine-caffeine combinations (look-alike drugs) in the 1980's. A decade ago, 13% of young adults and 3% of adolescents had tried cocaine [B*Ray, 1978]. Legal amphetamines were prescribed for 3-4% of all school children for "hyperactivity" in 1973. For obesity, 5 million prescriptions were written in 1976, a drop to 20% of the rate one decade earlier [B*Ray, 1978]. Caffeine use is so ubiquitous in beverages, over-the-counter remedies, and chocolate (as theobromine) that it is accepted as a non-drug by most people. Yet excess use has been associated with anxiety syndromes and sleep disorders [J*Greden, 1974]. Hyperactivity of children may be associated with caffeine/theobromine in

the diet. In 1976, caffeinated beverages were preferred by more than half of the US population above age 10 [B*Ray, 1978]. With recent attention to caffeine as a toxic agent, and more widespread availability of decaffeinated beverages, there may be a reduction of unintentional caffeine toxicity. However, 8% of Americans have used over-the-counter stimulants intentionally to extend or facilitate performance (e.g., long-distance drivers, students) or to reduce appetite.

Opioid drug use and abuse can be divided into medical and "street" subcategories, with a variety of different opioid drugs used individually or in combination [B*Schuckit, 1979]. Street heroin had been tried by 3% of inner-city residents in 1974, and was actively being used regularly by less than 0.5% [B*Chambers, 1977]. Dosage and regularity of use varies in response to purity of supplies, which in turn are usually affected by the efficacy of recent law-enforcement efforts to intercept illicit heroin shipments. When purity drops, prices rise, and those who are addicted may substitute other street or prescribed opioids (some may enter methadone maintenance programs), while new recruits may not become addicted at all [J*Zinberg & Jacobson, 1976]. The proportion of prescribed opioids who are misusing or abusing them has not been satisfactorily estimated, but probably many of those who take them over prolonged periods are dependent. Those with terminal illnesses such as cancer may suffer little of the negative effects of dependence and tolerance and none of the social consequences (i.e., stealing, violence, dirty needles) because their use is medically supervised. However, those with chronic non-terminal pain syndromes who are on opioids may benefit from alternative treatments (i.e., hypnosis, transcutaneous electrical nerve stimulation, biofeedback) and detoxification. In the previous century, opioid use and dependence was much more widespread than now, with unregulated supplies of opioids available in numerous patent medicines [B*Ray, 1978]. Typical users of that era were middle-class middle-aged caucasian housewives, where now the street heroin abusers are usually younger, often lower class, and more often minorities. Pharmaceutical opioid abusers may still demographically resemble the patent medicine abusers of the 19th century, except that health care professionals should be added, with a very high prevalence rate (see Chapter 9).

Tobacco products are still dominated by cigarettes, though cigars, pipes chewing tobacco and snuff are making come-backs among those wishing to quit smoking dangerous cigarettes. It must be remembered that cigarettes were a phenomenon of the last century only, and that previously chewing tobacco and snuff were preferred [B*Ray, 1978]. Demographic profiles have changed over recent years with female smokers overtaking male smokers in prevalence. Altogether about a third of all people above age 12 smoke. There has been greater interest in low tar and nicotine containing and filtered cigarettes, although it is not known how many people actually achieve the lower tar and nicotine absorption. Recent introduction of a nicotine-resin impregnated chewing gum for the purpose of nicotine detoxification of smokers [R*Merrell Dow] may help reduce the prevalence of smokers if the gum itself does not become an object of abuse. The prevalence of

problematic tobacco smoking is high, estimates being that 300,000 people die prematurely each year in the US as a consequence of smoking due to cardiovascular and lung diseases, and cancers [B*Rutledge, 1983]. In addition, excess morbidity includes peptic ulceration and neonatal toxicity [B*Meyer, 1983] in addition to the other disorders.

Marijuana use is very prevalent among young people in the US today, with over 25% using it regularly and 17% of those who have ever tried it smoking it daily [B*Ray, 1978]. Over 21% of all youth and adults had tried it in 1976, but the highest prevalence of experimenters were 53% of those aged 18-25 that year [B*Ray, 1978], who are now 26-33 years old. 6% of 12-13 year olds had tried marijuana that year, and half of these had smoked during the previous month. With such widespread use of marijuana in the population and reduced penalties for possession in many jurisdictions, it is apparent that marijuana is here to stay. Nevertheless, there has been a mild reduction of marijuana experimentation among youth more recently, with preference towards alcohol. The prevalence of problematic use of marijuana has not been estimated.

Other hallucinogens are used occasionally by probably 20% of youth, with a small minority of youth using them regularly [B*Schuckit, 1979]. Other inhalants had been tried by 20% of urban adolescent girls and 33% of their male counterparts in the early 1970's [B*Schuckit, 1979].

There is considerable variation from one study to another in prevalence rates, partly attributable to the different criteria used. For example, sociological studies often have used drinking quantity and frequency by self-report to sort people into drinking categories [B*Cahalan, Cisin & Crossley, 1969]. Yet, one cannot say that all heavy users are chemically dependent, nor conversely that every light user or abstainer is not chemically dependent. People shift drinking and drug use patterns over time in response to changes in availability, price, fashion, and personal circumstances [B*Vaillant, 1983]. In addition, some people seem able to consume high doses without developing harmful consequences (a necessary element of most definitions of substance abuse), while others develop substantial problems from lower doses. Infrequent binge-drinkers or drug abusers have a low average daily consumption, though they might have substantial trouble during or following most episodes of use. Some people have the compulsion to ingest a psychoactive substance but do not experience symptoms or signs of physiological withdrawal. These persons probably can be considered to have PSYCHOLOGICAL DEPENDENCE as opposed to PHYSICAL DEPENDENCE. For some people, the social pressure is the most important determinant of relapse, and for them it might be best to consider them SOCIALLY DEPENDENT on drugs. Some people combine normal doses of several potentiating drugs and would not seem to be deviant unless the entire pharmacological picture were analyzed. Multiple drug use is common and poly-drug abuse is more prevalent among psychiatric patients, those prohibited from using their favorite drug (e.g., those on methadone maintenance, those in stressful situations apart from

their social support system (e.g., young soldiers, prisoners), and the elderly who are often on many drugs from many doctors for their many symptoms or illnesses [B*Schuckit, 1979].

TEACHING HINTS: Epidemiology

The residents can read about the prevalence of alcoholism or other substance abuse in the general population, in the state or province of their practices, and in special populations for which they care (e.g., certain ethnic groups, occupational groups, and so on). Attention should be directed towards age and gender biases in the populations served, because clinic staffs do not always reflect the total population from which patients come [B*Cahalan, Cisin, and Crossley, 1969; B*DuPont, Goldstein & O'Donnell, 1979; B*Kalant & Kalant, 1971; B*Marvin et al., 1976; B*Newmeyer, 1978; B*Richards & Blevins, 1977; B*Rittenhouse, 1977].

One should also look at both the direct and indirect victims of chemical dependency: the children, spouses, and co-workers of substance abusers [B*Woodside, 1982]. Using the population estimates from the literature, one can project prevalence rates in the clinic or community by using practice demographics. If these are not on hand, they can be obtained by sampling the practice or by requesting them from local government.

One can calculate the expected prevalence of abstinence; light, moderate, and heavy drinking; alcoholism and other drug dependencies and or usage states (e.g., tranquilization, or marihuana or tobacco smoking) in one's patients and their family members. In a continuing seminar setting, this exercise could be shared among various residents. Then the residents can survey their own patients to see whether these projections are accurate.

Discussion after attempting this assignment could center around any of the following:

1. the difficulties in finding good local estimates of substance abuse prevalence;
2. the doubts of validity that arise when one is gathering information about the higher dose categories of substance use;
3. the under- or over-utilization of health-care services by certain groups (e.g., females, the poor) and the impact that might have on practice demographics and substance abuse prevalence projections;
4. the reasons for greater visibility in one's practice of indirect victims of alcoholism than alcoholics themselves;
5. the common signs and symptoms presented by the indirect victims of addictions; and
6. the differences among the rates generated by different residents attempting this exercise and the reasons for the differences.

EVALUATION: Epidemiology

How familiar is the resident with the current knowledge about the prevalence rates of the substance abuse disorders in the population being served by the clinic? Does the resident realize the variables which alter prevalence statistics from location to location, or from one subgroup to another within the clinic? Has the resident evaluated his or her own rate of diagnosis of substance abuse among patients for whom he or she is caring by comparing to the predicted prevalence rates? Has this led to any changes in practice?

CASE-FINDING

LEARNING OBJECTIVE: Case-finding

The resident must learn to screen for substance abuse problems. Early case-finding is preferable to waiting until the case has progressed substantially. The resident should have discovered screening procedures for the early detection of alcoholism and other addictive disorders which are cost-effective and fit into the style of practice planned by the resident.

COMMENTS: Case-finding

Family physicians are in an excellent position to accomplish early recognition and diagnosis of the addictions because of several key factors about their relationship with patients, their families, and the surrounding community:

1. Family doctors have a comprehensive view of illness and health unmatched by any narrow specialist.
2. Family doctors have a mandate to prevent unnecessary illness and consequential disabilities.
3. Family doctors have a commitment to coordination of health-care services that are delivered to the patient and family.
4. Family doctors see patients over an extended period of time, and through continuity of care, build upon prior contacts with the family to develop a longitudinal data base and interpersonal rapport.
5. Family doctors are interested in and trained to deal with the bio-psycho-social aspects of the human condition.
6. Family doctors stay in regular contact with not only the patient, but also with other members of the family and the community.
7. Family doctors are aware of the characteristics of the community in which the patient and family live and their relationship to the community.
8. Family doctors tolerate the ambiguities of medical reality that elude exactness.

Utilizing this unique combination of skills, attitudes and knowledge, family physicians can and should recognize cases of chemical dependence much earlier than most other physicians [B*Doherty & Baird, 1983; B*Liepman, 1984; J*Westermeyer, 1980].

A common attitude of most medical school graduates is that alcoholism is an uncommon condition, except in poor, inner-city, skid-row males [J*Liepman, Whitfield & Landeen]. Likewise, drug addicts are thought to be tough inner-city young people. These stereotypes are far from true, since chemical dependence is an equal-opportunity disease. Nevertheless, these stereotypes generate stigma which affects the patient, the family, and even the unwary physician. Many patients and families suffering from alcoholism or other chemical dependencies deny the existence of the illness or its severity largely to protect themselves from shame and embarrassment. They often mislead the physician by under-reporting their drinking or drug use and denying its etiologic contribution to its consequences. They may do this by neglecting to spontaneously mention use of psychoactive substances at times when the clinician feels it would have been relevant information, by evasively responding to questions from the clinician, by bending the facts about use, or by consciously lying. Two case examples follow:

CASE 1: 14 year old Joanne had been drinking heavily on weekends for about six months but knew that it was illegal and that her parents would not approve. She also doubted that her doctor would approve. She came here because she had missed her menstrual period for two consecutive months. She had been having unprotected intercourse with her boyfriend, with whom she had been doing the drinking. The clinician realized the importance of determining the risk of the fetal alcohol syndrome before embarking on the intervention with Joanne and her parents to deal with the pregnancy. Her initial guilt and fear caused her to deny that she drank at all. After hearing this, the clinician sensitively discussed drinking and drug use among her peers and inquired about her reasons to resist joining with them in party-making. He also explained his responsibility to protect her confidentiality, including the difficult bind that would arise if she wished him to conceal this information from her parents. He mentioned that the alcohol and drugs may have an effect on a pregnancy. After this discussion, she cried and told him about her drinking and marijuana smoking.

CASE 2: 81 year old Minnie, a widow of 8 years, had been appearing progressively more depressed on successive visits. She still lived alone in the family homestead. She reported increasing insomnia, loneliness, apathy towards living, anxiety, guilt, and mentioned death twice during the visit, though she was not diagnosed as having any terminal diseases. She had missed several recent appointments, but always called the nurse to reschedule and to get her medications renewed. She was

losing some weight, allegedly to get rid of her excess fat. Her family had expressed concern about Minnie during their health care visits, complaining that she was becoming less active in the community and was refusing contact with grandchildren and family members quite regularly. Minnie was reluctant to discuss her drinking and use of her sleeping pills. She acted somewhat indignant towards her physician for asking about them. She admitted to taking a "night-cap" regularly and that she had this with her regular sleeping pill lately because of her insomnia. On further questioning, she admitted that she had also been taking some over-the-counter (OTC) sleep-aids which she found in the medicine cabinet. She only admitted the extent of her drinking when the physician sensitively inquired about the quantity of alcohol she purchased each week. When the one drink per night did not jive with two bottles of sherry per week, he inquired about the time the night-cap began and the size of the container, discovering that she began drinking at 7 pm and drank from a large mug. She then tearfully admitted that she had bought additional supplies of her OTC sleep-aids several times. She admitted that she was becoming concerned with the escalation of doses of the sleeping pills and had worried about what might happen with concurrent drinking (although she was not altogether frightened by the prospect of dying by drug overdose).

The unfortunate tendency for chemically dependent patients to mislead their doctors can lead to misinterpretation of subtle alcohol/drug abuse related symptoms and signs of ill health in the patient and family, which could signal early the need for medical intervention. Failing to recognize the role of addictive disorders in such illness facilitates physician benign neglect of serious health problems until they progress to later stages, defeating the health-promotion goal of family practice. It also tends to foster family and patient acceptance of the alcohol-related consequences as inevitable and unrelenting life events over which they have no option of control. Unless the physician is able to break through the stigma and denial barriers, they may interfere with gathering an honest data base from the patient and other family members.

It is important to understand the motivation of patient and family to mislead the physician is usually not malicious, rather it is the secondary consequence of denial to protect oneself from facing the unpleasant reality. Minnie did not have to admit to herself the extent of her tolerance by drinking only one night-cap per night, hence she fooled herself, and indirectly, her doctor. Minnie also did not realize that concurrent ingestion of multiple sedatives had a multiplicative effect, so she could appear (to herself and others) not to be drinking to excess, though the drinking was certainly increasing.

In a systematic review of the database in medical records of a university primary care clinic, very few records revealed any

information about substance use, revealing neither a positive nor a negative history [B*Liepman, 1983 & 1984]. While one would presume that primary care clinicians usually gather this data, the lack of entries in the chart suggests that useful information was not gathered. In another study of primary care clinicians, the Michigan Alcoholism Screening Test (MAST) [J*Selzer, 1971] was administered to all patients in the waiting room and then placed on the record with a guide for interpreting the results. In less than 20% of the records was there evidence that the resident used the information to initiate treatment [J*Breitenbucher, 1976].

It is important that the doctor constantly maintain a high index of suspicion that even patients with a negative history might develop addictions. This will aid in recognizing subtle clues that slip through the defensive armor of the patients. A more complete data base can then be generated by applying the supportive confrontation techniques outlined in Chapter 4.

SCREENING

The practice of family medicine is a combination of focused, problem-oriented episodes of care and preventive health screening maneuvers applied periodically to maximize early detection of disease. With the addictive disorders being both common and destructive, it is incumbent upon family physicians to regularly screen for them [B*Anonymous, AMA, 1973; J*Lewis, 1980; B*Liepman, 1983 & 1984; J*Martin, 1980; J*Tennant, Day & Ungerleider, 1979]. Screening may be done by history, questionnaires, physical exam or laboratory tests. Simple questions such as the CAGE [J*Ewing, 1978; J*Mayfield, McLeod & Hall, 1974] or just casually asking the patient about substance use may pick up many cases that would otherwise have been missed until much later. Formal questionnaires such as the MAST may be administered as part of routine health maintenance visits [J*Selzer, 1971]. The MAST has a very high sensitivity (95%), hence it is rarely fooled, even by an alcoholic with lots of denial. There are many other questionnaires that are available [B*Huba, Bentler & Newcomb, 1981; B*Jacobson, 1976; B*Knox, 1982; J*McAuley, Longabaugh & Gross, 1978; J*Swenson & Morse, 1975] that may be used to increase identification of alcohol and drug problems.

Physical examinations may reveal signs of alcoholism. Early signs might include vascular hypertension [J*Klatsky, Friedman & Siegelau, 1974 & 1979] with mild tachycardia, fine tremor, the odor of alcohol on the breath, unexplained or poorly cared for injuries [J*Israel et al., 1980], or enlargement and tenderness of the liver. The later signs include many of the complications of alcoholism. For intravenous drug users, signs might include needle tracks, sometimes in cleverly concealed locations, heart murmurs, nasal septal perforation, skin pitting, and pupillary changes. A review of the medical consequences can be found in [B*Liepman, 1984; J*Merz, 1974; J*Pell & D'Alonso, 1968; B*Lowenfels, 1982; B*Wartenberg & Liepman, 1984].

During hospital care, routine laboratory tests are done some of which may help alert the physician to the diagnosis of suspected alcoholism [J*Quinn & Johnston, 1976; J*Shaw et al., 1979; J*Westermeyer, Boheny & Stone, 1978]. Elevations of the GOT or the MCV can signal alcoholism with 85% certainty [B*Drum & Jankowski, 1977 & 1982]. Elevation of the GGT is even more sensitive, though less specific, but the GGT should not be elevated in healthy non-pregnant individuals [J*Augustine, 1967; J*Casey et al., 1975; J*Goldberg & Martin, 1975; J*Konttinen, Hartel & Louhija, 1970; J*Lamy et al., 1974 & 1975; J*Lindenbaum, 1980; J*Rosalki & Rau, 1972; J*Shaw & Lieber, 1980; J*Wallerstedt & Olsson, 1978; J*Whitehead, Clarke & Whitfield, 1978; J*Zein & Discombe, 1970]. Identification of other serum abnormalities such as elevation of HDL [J*Hartung et al., 1983; J*Haskell et al., 1984; J*Mendelson & Mello, 1973] or a far higher uric acid (though perhaps within normal range) at admission in contrast to later in the hospitalization may also lead one to consider proceeding with a diagnostic work-up for alcoholism. Quantitative results from a breathalyzer or blood sample when one smells the odor of alcohol on the breath may reveal heavy intake and tolerance [B*Anonymous, 1973; J*Clark et al., 1979; J*Ewing, 1978; J*Lewis, 1980; J*Senior & Sloan, 1981]. Results of a drug abuse toxicology screen, usually performed on urine, can also verify history or identify occult drug ingestion.¹

TEACHING HINTS: Case-finding

Have the residents review the methods for identifying cases from among a population of patients. Have them report to one another on the different methods which have been suggested focussing on practicality and appropriateness for hospitalised or ambulatory patients.

In some cases, trial exercises to utilize these approaches might be included in the assignment on a sample of their own patients for an interval of time. (e.g., one resident might administer the family alcoholism questionnaire to all his or her patients for a week; another might take a thorough alcohol history from all inpatients with elevated SGOT or MCV values). At the end of the interval, have them review their results together as a group, comparing the rates of sensitivity and specificity of each of the tests they selected.

¹ Be certain what the lab can detect before counting on it to help you make a diagnosis. Some labs cannot detect certain drugs or combinations or levels. For example, phencyclidine levels that are clinically toxic to a moderate degree can only be detected by high-pressure liquid chromatography techniques; some labs will report negative results on this test when the sensitivity of their technique is rather low.

EVALUATION: Case-finding

The resident's patient population will be the test of case-finding skills. By examining medical records or asking the resident to tally all diagnoses of substance abuse problems from among his or her patients, the teacher can quantitatively assess the thoroughness of case-finding by calculating the fraction of the predicted² cases which are found (diagnostic sensitivity). The proportion of cases which are in the early stages in contrast to late stages will provide information about the level of the resident's index of suspicion. While precepting, the style of assessment may be examined. Watch for patient alienation by insensitive questioning, resident collusion with patient denial, lack of thoroughness or persistence in history taking, or failure to assess certain patient groups who do not fit the mythical stereotypes of alcoholics or drug addicts. In a classroom setting, the student's skills at gathering substance use history may be evaluated by role play procedures either using instructors, other students, or AA and Alanon members who have volunteered to pretend to be patients. Video tapes made of the sample data base collection can be useful in careful evaluation. It is best to determine at the outset what criteria for assessment of the history gathering skills will be used. If these can be shared with the students before teaching them, there will be more cooperation and achievement of goals.

DIAGNOSIS**LEARNING OBJECTIVE: Diagnosis**

The resident will learn how to interpret historical, physical exam and laboratory data in such a way as to diagnose chemical dependencies or rule them out.

COMMENTS: Diagnosis

The diagnostic process for the addictive disorders has not been easy to teach or learn because of controversy and lack of standardized criteria. However, this is currently improving. The American Psychiatric Association has recently disseminated a set of guidelines for the diagnosis of the substance use disorders [B*APA, 1980] which are fairly specific and useful. For most of the psychoactive substances there are two options: abuse or dependence. It is possible to specify each drug class and multiple substance use disorders. Prior to this, the National Council on Alcoholism Criteria Committee had published guidelines for alcoholism [J*National Council on Alcoholism, 1972], but these were

² based on epidemiological estimates or compared to the best substance abuse diagnostician in the practice

cumbersome and less specific. Clearly, one of the main problems with diagnosing the addictive disorders has been the wide individual variation in terms of patterns of use and the quantitative and qualitative accumulation of harmful consequences [B*Rohan, 1982]. Attempts to make a diagnosis based on norms of quantitative ingestion fails on the basis of differences in body size, rate of absorption, distribution pattern, rate of elimination, tolerance/cross-tolerance and concurrent drug use. Sociocultural differences in normative behavior also alter the social impact of any pattern of ingestion, depending on the context in which this pattern is found. Attempts to diagnose based on consequences also fails if particular consequences are singled out, because patients vary in their biological, psychological and social vulnerabilities (for instance, a Mormon would develop social problems with the first drink, while an Irish-American would find acceptance of quite a large amount of drinking; some people have a greater hereditary vulnerability to developing the Wernicke-Korsakoff disorder). Efforts to identify the "alcoholic or addictive personality" have also failed to identify unique personality profiles or factors which will identify alcoholism or other addictive disorders, although there are identifiable scales on the MMPI (depression & psychopathic deviance scales) which seem to stand out in drinking alcoholics (but they disappear with recovery) [B*Jacobson, 1976; B*Knox, 1982]. The most successful efforts seem to be those which focus on the overall accumulation of harm.

The key elements in a DSM-III diagnosis of abuse is chronic (more than one month) pathological (socially or occupationally harmful) pattern of drinking or drug ingestion. By adding the biological phenomena of either tolerance or withdrawal, the patient qualifies for dependence [B*Robins, 1982]. Since some drugs do not lead to tolerance or withdrawal syndromes, use of these drugs can only be diagnosed as abuse. The old terminology of physical dependence seems to have been equated with dependence in DSM-III, and abuse encompasses psychological dependence. The use of social and occupational measures of harm attempts to avoid the traditional later stage medical signs like cirrhosis or peripheral neuropathy, assuming correctly that most of these illnesses do disrupt social or occupational function if they have not already been disrupted earlier by more subtle factors. The result of this diagnostic schema is to demand of the diagnostician more attention to the social functioning of their patients than was often paid in the past by medical specialists. The family physician may not notice much difference, since attention to social factors is fairly standard operating procedure. Subtle signs of social dysfunction may include memory problems (alcoholic amnesia), moodiness, marital or parenting problems, sexual dysfunction, financial problems, poor coping with stress, irresponsibility, unreliability, erratic performance. The main problem with this list is not identification of the items, but rather attribution of these signs to the drinking or drug use, since many patients will place cart before horse in blaming their substance abuse on stress or problems they have encountered.

One useful clue to the diagnosis is whether the patient is in control of substance ingestion. At first look, some may be obviously out of control, while others may take more scrutiny before a decision can be made. One need not be out of control all the time to be out of control enough to accumulate repeated harmful consequences. Some people will lose control of substance ingestion only episodically, such as the woman who drinks alcoholically only on major holidays, or the man who binges on cocaine only on payday. Many people construct "control rituals" [J*Zinberg & Jacobson, 1976] which they use to prevent losing control (e.g., "I always leave my wallet in my glove compartment when I go into a bar, taking only a few dollars with me," or "I avoid shooting heroin when I am depressed"). Others will encourage, manipulate or accept external controls on their behavior when internal controls fail (e.g., "My wife gives me my disulfiram every morning before breakfast," or "I asked the bartender to offer me no credit and to serve me a soda whenever someone buys me a drink"). If and when these internal or external controls break down, the substance ingestion gets out of control (relapse). Gathering information about control rituals, their efficacy and their occasional failures, may help the clinician to make a diagnosis in more subtle cases. Initial patient denial of loss of control, especially in males, may signal subconscious sensitivity to this "weakness of will-power" and may be perceived as an affront to self-esteem. But asking a patient whether thoughts of quitting or cutting down on substance ingestion had ever hit his or her mind may unearth the desired information or defensive chatter, either of which can be useful diagnostically. Loss of control over intake may affect the schedule of use and/or the dosages (e.g., "I never drink during the week, to preserve my image at work, but last Sunday I didn't slow down in time to be ready to go to work on Monday," or conversely, "I usually smoke no more than two joints per day, but during exam week, I finished off 100 joints!").

Information about exact frequencies and doses of intake, routes of administration, sources and purity of drug, and other details may be useful in deciding upon treatment approaches, but contribute little to the diagnosis. Likewise, detailed information about consequences, though useful in motivational counseling, will not contribute significantly to the diagnostic process, once it has been established that the person uses abusable substances in a way that causes repeated harm.

TEACHING HINTS: Diagnosis

Have the residents read DSM-III or other articles about diagnosis [B*Anonymous, 1973; J*Augustine, 1967; B*Bissell, 1980; J*Breitenbucher, 1976; J*Bulmer, 1980; J*Casey et al., 1975; J*Clark et al., 1979; J*Davis, 1980; B*DuPont, Goldstein & O'Donnell, 1979; J*Ewing, 1972 & 1978; J*Filstead, Goby & Bradley, 1976; J*Fink, Knott & Beard, 1974; J*Glass, 1977; B*Hofmann & Hofmann, 1975; B*Jacobson, 1976; B*Jellinek, 1960; J*Jones, 1979; B*Kalant & Kalant, 1971; B*Knott, Beard & Fischer, 1972; J*Knott, Fink & Beard, 1974; J*Knott, Thomson & Beard, 1971; J*Lewis,

1980; B*Liepman, 1984; B*Maletta, 1982; J*Martin, 1980; J*Mayfield, McLeod & Hall, 1974; J*McAuley, Longabaugh & Cross, 1976; J*Mezey, 1974; B*Mullin, 1978; J*National Council on Alcoholism Criteria Committee., 1972; B*Polan, Lea & Vibe, 1979, Chapter 4; B*Robins, 1982; B*Schuckit, 1977; J*Selzer, 1971; J*Silberfarb, 1976; J*Skynner et al., 1980; J*Smith, 1984; B*Sobell, & Sobell, 1978, Chapter 3; J*Sokol, Miller & Reed, 1980; J*Swenson & Morse, 1975; J*Tennant, Day & Ungerleider, 1979; J*Weinberg, 1974; B*Westermeyer, 1976; J*Westermeyer, 1980; B*Whitfield, Liepman & Williams, in press; B*WHO, 1964; B*Wilford, 1981; B*Zimberg, 1982]. Have them develop a diagnostic procedure that they could use with patients. Have them watch a film on making a diagnosis [A*Alcoholism: A Chronic, Treatable Disease; A*Alcoholism and the physician; A*Alcoholism: Early Diagnosis and Management; A*Clinical aspects of alcoholism; A*Decriminalization of alcohol abuse; Intake procedures; A*Doctor, you've been lied to; A*Liepman; A*Substance abuse and the physician; A*The physician's role: The diagnosis and management of alcoholism and alcohol-related disorders]. Then have the residents role play taking a pharmacologic and addiction history, using either each other, an instructor, or a recovering substance abuser volunteer to pretend to be a patient with an addiction. Usually a group of 5-6 residents can rotate the interviewer role, each spending five minutes gathering additional history that others prior have missed. Between rotations of the interviewer, the other residents can provide feedback on what was obtained and how it was done.

One way to help the learner to remember all the elements of an alcohol/drug history is through a mnemonic: CHEMICALS. Each letter stands for a topic of history that should be obtained [B*Liepman, 1984]. Briefly summarized, the letters stand for:

- C** Complications/Consequences of use or abuse
- H** Help sought for these problems in the past and outcome of that help
- E** Enablers in the Environment and their Effect on the illness; what Effect has it had on them?
- M** Maximum dose ever taken & Minimum dose it takes to get any effect: a Measure of tolerance
- I** Intake or Ingestion details: drugs, doses, frequencies, sources, routes
- C** Concerns the person has had about this problem in ~~past~~; thoughts of Changing
- A** Abstinence syndrome with sudden cessation; Advantages of Abstinence
- L** Losses if the person quits; disadvantages of abstinence
- S** Synergy between alcohol/drugs and other medications or illnesses

Have the residents use this schema to evaluate their own history gathering skills and those of their peers during role play exercises. They may also use it for awhile to record the clinical history on the medical record.

Patient management problems can pose diagnostic questions of graded complexity. The teacher might make up several and then use them with residents as a means of challenging their diagnostic skills. Or have the residents try to find a specified number of chemically dependent patients from among those for whom they care in the clinic or hospital.

EVALUATION: Diagnosis

The best way to evaluate the diagnostic skills of a family medicine resident is to observe the process in vivo. The in-service training exam [R*Corley & Fisher] may provide one way to do this in a standard fashion for all residents in the program. It may be done with real patients in the clinic or hospital. Or a simulated patient experience can be used to evaluate the diagnostic process. Watch for database acquisition problems, failure to properly interpret data that was obtained, and lack of understanding of the diagnostic criteria. If there seems to be a blockage to learning about diagnosis, one should explore personal attitudes, because the resident may be personalizing this lesson and may find it hard to accept the criteria being offered on the grounds of personal or family involvement with substance abuse.

STAGES OF ADDICTIONS

LEARNING OBJECTIVE: Staging the illness

The family physician should be able to judge the stage of the addictive illness so as to recommend stage-appropriate treatment components, and to counsel the patient and family as to the severity and extent of the disease progression that has occurred thus far.

COMMENTS: Staging the illness

Like all progressive chronic illnesses, the victims of the chemical dependencies can be arbitrarily sorted into groups by stage of the illness. Such a grouping has prognostic and treatment planning implications. Earlier cases usually have less resource loss (hence less grief and more support) than late cases. The recovery rate of very late stage cases (e.g., skid-row alcoholics) is quite low in contrast to earlier cases. By learning to determine stages, the student will have standards for diagnosis for each stage. This will prevent discarding one's suspicions that a patient has early chemical dependency because it hardly resembles an advanced stage case. This facilitates early intervention. It encourages the resident to approach the early case with therapeutic enthusiasm rather than to deny its existence until it progresses substantially. A suggested schema of stages is listed in Table 3-1:

Table 3-1: Stages of Chemical Dependence

STAGE	DESCRIPTION
Abstinence	Person has not begun to use drug, but attitudes are developing
Non-problem use	No negative consequences of use
High-risk use	Use is frequent, heavy, or usage patterns are dangerous
Problem use (or abuse)	First negative consequences arise from usage patterns
Chemical dependence: Early stage	Reversible, less serious negative consequences or threats do not motivate corrective adjustment of usage patterns
Chemical dependence: Middle stage	Irreversible negative consequences of use do not motivate significant corrective adjustment of usage patterns
Chemical dependence: Late stage	Multiple, serious irreversible negative consequences have failed to motivate corrective adjustment of usage patterns

TEACHING HINTS: Staging the illness

A series of case vignettes (real or fabricated) or films can be presented in a seminar setting for the students to determine the stage of the illness [A*A slight drinking problem; A*Alcoholism; A*Cause the effect; A*Doctor; A*Drinkers; A*Feelin' no pain; A*Francesca baby; A*Gale is dead; A*Gallant, 1979; A*If you loved me; A*I'll quit tomorrow; A*Lisa; A*Lots of kids like us; A*One day; A*Our brother's keeper; A*Rouk, 1982; A*Soft; A*Some; A*The enabler; A*The physician's role, parts 2 & 3; A*The secret love of Sandra Blain]. Residents might also discuss the differences in the treatment needs of patients in different stages of dependence (e.g., the differences between the case of an adolescent with a broken arm caused by combining drinking and rollerskating, and the case of the teenager's adult onset diabetic father, who is suffering from a second bout of alcoholic pancreatitis).

In the clinic, the resident might determine the stage of 10 consecutive patients suffering from alcoholism, or 20 users of

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other substances. If it is early in the residency, the resident might keep track of the treatment outcome of these patients to verify the different treatment needs and prognosis.

EVALUATION: Staging the illness

Have the residents include information about stages in their notes in the medical record and in case discussions during precepting. Compare your impression of the stage with that suggested by the resident. Ask for evidence if the your impressions do not agree.

CHAPTER 4: MOTIVATING CHANGE

By J.V. Fisher, C.L. Whitfield & M.R. Liepman

OVERALL OBJECTIVES

The family medicine resident needs to learn and utilize effective methods to alter the motivations of patients who are pursuing a dangerous set of behaviors as part of the clinical course of an addictive illness. In order to use confrontation and coercive approaches, the resident will need to examine ethical issues and to have developed attitudes that will support these clinical activities.

SUPPORTIVE CONFRONTATION

LEARNING OBJECTIVES: Supportive Confrontation

The resident should be able to obtain an adequate data base to determine the level of patient and family awareness of the chemical dependency and their degree of motivation to recover. The resident should be able to recognize supports and deterrents to successful recovery in the family and community. The resident should develop relative comfort in a variety of supportive confrontational techniques and exhibit tolerance of immediate failure of confrontation without giving up the effort to motivate recovery. The resident should learn to optimally match the confrontational technique to the patient.

COMMENTS: Supportive Confrontation

Patients who suffer from alcohol or other drug dependence often present to the family physician with a problem other than the chemical dependence. If the doctor recognizes the underlying cause but fails to confront the patient with this fact, the patient will not receive proper treatment. Yet, reluctance to confront the patient is all too common among residents [J*Breitenbucher, 1976; J*Fisher, Fisher & Mason, 1976; Strachan, 1968]. The process of confrontation must be done constructively to maintain the patient-physician relationship. Likewise, physician support of the patient and family must not facilitate progression of a serious, fatal disease process. Successful supportive confrontation requires knowledge and skills, as well as positive attitudes.

The goals of successful confrontation are: (1) the patient and family realize that they have an illness related to the problematic use of alcohol or other drugs; (2) the patient and family sincerely want to do something constructive to solve the problem; and (3) the patient and family commit themselves to in-

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vesting effort in a recovery program and engage in a therapeutic alliance with a skilled professional team.

ATTITUDES

In establishing a caring relationship with patients, one must make clinical rather than moral judgements, convey consistently a feeling of warmth, resist being manipulated or seduced by sick patient behavior, and convey optimism that treatment really can help. The key attitudes that facilitate effective therapeutic confrontation of chemically dependent patients are: (1) considering the patients as victims of a pernicious illness rather than as morally derelict persons; (2) believing that addictive diseases are chronic rather than acute diseases; and (3) believing that recovery is possible and that the family physician has a legitimate role and responsibility to facilitate recovery and healing.

In order to accomplish the foregoing, the confronting physician must be aware of his or her own feelings. In some cases, physicians are not even conscious of their attitudes, perhaps transferring anger at an alcoholic relative or personal friend upon their patients with alcoholism. The physician must be able to deal with the patient's frustrating behavior (i.e., the chronic relapsing nature of the addictive disease, patient resistance to therapeutic interventions) and have the maturity and equanimity to meet with denial and rebuff to confrontation efforts just as one deals with frustration when a medication or surgical procedure fails to cure an illness. The physician must even anticipate this as a normal phase in the confrontation process. Being able to interpret frustrating patient behavior as a symptom of a behavioral disorder often helps to redirect anger away from the patient-victim and towards the disease process. Then the frustrating behavior becomes a challenge to overcome rather than an assault on the physician's healing power and sincere desire to help patients. As family physicians mature, they learn to live with chronicity, to accept goals that fall short of cure, and to accept these realities without bitterness. One must realize that curing the patient may not be possible, but that caring for the patient and family is always an honorable option.

KNOWLEDGE

It is important for the physician to understand the illness, its natural course, and its treatment before delivering "bad news" about it to the patient. Equally important, the physician needs a well-thought-out conceptual framework, including diagnostic criteria and familiarity with the basic features of addictions, in order to inform the patient and family intelligently about the illness and its impact. This information can be obtained from numerous sources [B*Catanzaro, 1968; B*Gitlow & Peyser, 1980; B*Johnson, 1973; B*Pattison & Kaufman, 1982; B*Whitfield, Liepman, & Williams, in press; B*Zimberg, 1982].

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TABLE 4-1: Deterrents to Successful Confrontation

CHARACTERISTIC	PATIENT	DOCTOR
Ignorance	of disease chemical dependence and its pervasive harmful consequences	how narrow & biased is one's perception of the illness & the victim (i.e., stereotypes)
Fear	of rejection by others	of rejection by patient
Fear	of being incurable and hopeless	of being incapable of managing the illness
Fear	of losing advantages of the chemical dependency	of losing the reason for the patient to visit
Anger	challenging the doctor or passive-aggression	disinterest in patient; punitive or judgmental behavior
Rationalisation	alibis for substance abuse & consequences	accepting alibis or making excuses for the patient's need to abuse
Projection	blaming others for substance abuse & consequences	blaming patient for frustration & anger at difficulty engaging the patient in recovery program
Impatience	desire for instant relief of consequences or recovery	to get patient into recovery program and relieve worry about impending harm
Reluctance	to begin recovery and give up chemicals	to confront patient and risk alienation of patient
Denial	wish to avoid having to change & accept stigma	wish to avoid conflict & stigmatizing patient & self

The family physician should know the defensive deterrents that patients and families use to deflect concerned people who try to encourage them to seek help (see Table 4-1). Likewise, the doctor should be aware of strategies that can be used to respond effectively to the psychic and social needs which triggered the defensive strategies of the patients and families.

It is also important for the physician to realize the difference among various times in the family life-cycle, in terms of readiness for change. Smilkstein [J*1980] proposed a model for

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understanding why stressful times, family crises, and life-stage transitions seem to be better opportunities to confront chronic unhealthy behavior than during quiescent, stable periods of life.

The family physician should be aware of opportunities to use for leverage to motivate recovery. For instance, patient requests for documentation of physical health (e.g., to return to work after absence, for insurance, to tackle a special job) become opportunities to confront the patient with unhealthy behavior patterns. Knowledge that a patient's employer has an organized employee assistance program and health insurance coverage for substance abuse care would help counteract deterrents to recovery.

SKILLS

The first prerequisite to confrontation is the ability to acquire an adequate data base that meets acceptable diagnostic criteria [B*Anonymous, 1973; J*Clark et al., 1979; J*Lewis, 1980; A*Liepman, 1978; B*Liepman, 1984; J*NCA, 1972; J*Weinberg, 1974]. Without such a data base, confrontation proceeds on untenable ground, and the patient may very well challenge the diagnosis (see Chapter 3 & 8). The more diagnostic evidence that can be compiled, the greater the likelihood of a successful outcome of confrontation.

The person with alcoholism or other drug dependence is hyperperceptive and acutely aware of those signals from the physician (verbal or nonverbal) that the patient perceives as moralistic or rejecting. Such an interpretation may doom any confrontation endeavors by weakening the patient's trust in the physician's objectivity and nonjudgmental approach to medicine.

Confrontation efforts are best made after the physician has established a trusting relationship with the patient. Unless an emergency presents itself, the family physician has the opportunity, because of the ongoing relationship with the patient, to prepare incrementally for the confrontation.

The ability to select the appropriate time, the "critical moment," to present the confrontation, is a clinical skill and more intuitive than cognitive in its nature. The physician is faced with the problem of confronting a problem either too soon or too late. If confrontation is done too soon (before a relationship has been established and adequate diagnostic evidence has been secured), the patient may reject both the diagnosis and the physician. Lacking an authentic, irrefutable data base, the physician may convey diagnostic ambivalence which the patient and family may use to reinforce the denial system. The patient may correctly interpret this as an example of judgemental pseudodiagnosis which he or she may have previously encountered from friends and family many times before. Confrontation when the patient is satisfied with life and appears devoid of "pain" may damage the physician-patient relationship if adequate data to support the physician's concern cannot be brought forth. Clearly,

it is better to confront a patient during a crisis, if the data base is secure enough to support the physician's contentions [J*Smilkstein, 1980]. However, an inordinate delay in confrontation (e.g., waiting for years for the "right moment" can be considered enabling behavior [J*Levinson & Ashenberg-Straussner, 1978]). If the physician waits too long, the patient or family may interpret this delay to indicate uncertainty about the diagnosis, lack of danger, cowardice about confronting the patient, or a desire to facilitate the damaging progression of the illness. Failure to choose the proper time to confront can weaken, poison, or terminate the physician-patient or physician-family relationship.

The approach to confrontation must be individualized. The physician should have a repertoire of tactics. If the patient trusts the doctor and if the doctor understands the patient as a person, the intervention tactics can be tailored to the personality and circumstances of the patient. This statement assumes that the physician can anticipate the probable outcome of the efforts, and is prepared to deal with them (e.g., if rationalization is expected, he or she may have the skill to deal with this behavior).

Some tactical approaches that can be employed are:

INTERROGATION: "Have you ever wondered whether some of your problems could be related to your drinking?" By asking the patient or family to self-examine, they are likely to "own" the results of the search.

DECLARATION: "What you have told me and what I found on your physical examination and laboratory tests indicates that your problems are a direct result of drinking." This is most useful for those who value the authority of the physician no matter what is said.

SELF-IDENTIFICATION: Introduce the patient and family to people who are recovering or offer them books or movies on the illness which are likely to grab them affectively. If they can identify with what they see, hear and feel, they may begin to accept the diagnosis. This is particularly useful for those who have low self-esteem which is threatened by the diagnosis.

INCREMENTAL INSIGHT: Over a series of visits with the patient and family, present segments of data that may lead to some insight on the patient's part (although the patient may be reluctant to admit it) and prepare them for the next intervention step (e.g., first, notice tender liver and draw blood for liver enzyme studies; second, report elevation of serum liver enzymes and wonder about possible causes such as cancer, parasites, and toxic or physical injury--order scan or

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ultrasound or liver biopsy; third, report result of further studies and inquire in detail about toxic exposure--suggest repeat enzyme studies after trial abstinence; fourth, report drop in enzymes after abstinence or discover failure to abstain despite promise). Like a detective mystery story, the patient and family get drawn into the pursuit of etiology of the problem, fueled by anxiety and concern. Hence, they do not take the final outcome lightly.

EDUCATION. This approach can be utilized with those who deal with their problem at the intellectual level or who rely on rationalization to sustain their denial (e.g., "I can't be an alcoholic--I only drink beer.").

Patient and family education at some point in the intervention process is a must (see Chapter 8). So much ignorance exists about the genesis, evolution, chronic relapsing nature of the illness, and its pervasive effects on all areas of life, that it becomes the basis of behavioral mistakes by patients and families (e.g., accepting sleeping pills for alcoholic insomnia, or blaming the alcoholic for having "caught" the disease). This ignorance breeds fear and despair, which must be dispelled by valid factual and dispassionate information. It must be conveyed to the patient and family that this is a treatable illness, because often hope of any improvement has been forsaken. People with alcoholism or other drug dependence should be told they are not responsible for having caught their illness--but only if they accept responsibility to obtain and utilize treatment resources if recovery possible.

The skillful physician is aware that intervention efforts may well meet with defensive denial, hostility, ambivalence, rationalization, or projection. The patient needs these defenses to maintain stability in the face of the therapeutic confrontation that has change (rather than stability) as its goal. Hence, the doctor must deal constructively with these defense mechanisms and the patient's affect by tactics to overcome them without seriously harming the patient or their relationship, just as a surgeon must overcome bodily defenses (i.e., pain, muscle spasm) in order to change anatomy. The method of the opposites can be an effective foil to these behaviors:

1. Ambivalence can be met with firmness;
2. hostility by an overly-friendly attitude;
3. and denial by overacceptance.

Dealing with the patient's affect is vital to the doctor-patient relationship. Acknowledgement and acceptance of the patient's affect and asking for explanation of it, though perhaps uncomfortable to the patient, may put the therapeutic alliance on a firm basis of trust and understanding.

Despite all the preceding suggestions for dealing with predictable outcomes of intervention, and because of the apparent impregnable denial system the patient has erected, initial intervention efforts may not be successful. Do the physicians consider this rejection as a personal affront to their wisdom and integrity? Do they interpret this one rebuff as a failure, and thereafter abandon all further efforts to intervene? Family physicians have unique opportunities that may help them to define their responsibilities in intervention efforts. The family physician has the responsibility to be persistent and continue efforts to form a therapeutic alliance to gain support for commitment to a treatment program. The ongoing interactions between the patient and the family physician form a relationship that other specialists do not have. Additionally, and equally important, the access that the family physician has to all members of the family, in health and disease, may mean that family members can be enlisted in intervention efforts and can be encouraged and supported to take care of themselves.

If the family physician offers a program with optional therapeutic possibilities, and the patient finds reasons for not selecting any of these opportunities, the family physician may well question the patient's sincerity and lack of motivation, and may choose to confront the patient at this point with this observation.

What if the confrontation fails? What may initially appear to be a failure of confrontation (when the patient seems enmeshed in the denial stage of the illness) may have to be assessed differently when considering the long-term effects of the effort. The salience of alcohol dependency (and the alcoholic's imperative need to maintain the bulwark of denial) often make the initial attempt to intervene appear unsuccessful. The patient may return to visit the family physician for reasons other than alcohol- or drug-related problems. These visits give the family physician another chance to inquire about these problems and again attempt to intervene.

Even if the patient never returns to see this doctor, the patient may still realize, in more lucid moments, that a caring physician who speaks with knowledge and authority has explained that the patient has the disease of chemical dependence, which is treatable, and that help should be sought. This germinal truth, planted by the family physician, may then eventually bring, or force, the person with alcoholism into a treatment program.

CRISIS INTERVENTION

A natural crisis may be an opportunity to motivate the chemically dependent patient or family to seek treatment for the drug dependence. If the family physician is ready to refer the patient as soon as the crisis occurs, the family may remain open-minded long enough to begin to change. But if the doctor waits until the crisis has passed, probably the family will forgo the opportunity and choose the status quo (see page 8-29).

PEER COERCION

Enlisting the family and social support network as allies in motivating recovery can be a potent force and valuable asset to the concerned clinician. If a member of the social support network actively seeks help for the chemically dependent person, for themselves, or for others in the network whose emotional or physical health seems in jeopardy, they may be open to forming a therapeutic alliance that may lead to successful confrontation of the chemical dependence. This person is called the "emissary" [B*Liepman, Wolper & Vazquez, 1982]. In other cases, there is no emissary, but the physician may utilize opportunities to meet with and discuss the case with others during the regular course of his or her practice (e.g., when the family comes in with the patient, a family visit can be held and the issues can be discussed without violating confidentiality [J*Anonymous, 1972]).

A family/social network systems treatment approach has evolved called the "INTERVENTION" [B*Johnson, 1973] or the Family Constructive Coercive Confrontation (FCCC) [B*Liepman, Wolper & Vazquez, 1982] which can be used to motivate change in a social system which has been resisting recovery and may or may not be currently in crisis. If not in crisis, the first step may be the family physician speaking with family members about concerns he or she might have about the substance abuse problem. If the family is in distress, the FCCC may begin with a request for help of some sort from an emissary (e.g., "I can't sleep at night because I worry about my son who stays out all night with his friends" or "I think my daughter is pregnant"). When the emissary is a child, frequently he or she is brought to the attention of the community authorities because of some sort of behavior problem. The helping professional assesses the situation, trying to determine (a) who is involved, (b) whether chemical dependence is a factor, (c) what kind of treatment is needed, and (d) who among the social network can help motivate entry into treatment. Then, the emissary is told about the FCCC procedure and is encouraged to recruit others from the social network to join the team in FCCC training and team-building exercises.

If the emissary succeeds in bringing in several other enablers from among the extended family, close friends, supervisors, clergy, the doctor, and others to an organizational meeting of the FCCC team, they are shown movies [A*The Enabler, A*The Intervention, or A*I'll Quit Tomorrow] and trained in the basics of chemical dependence and gentle constructive confrontation. They may meet several times to clarify and express to one another their concerns about the drinking or other drug use. Each team member prepares, for a group presentation, a report on personal concerns about the chemical dependency behavior. They practice presenting this to the chemically dependent person in a firm but supportive fashion with the hope that the presentation will overcome patient denial and motivate immediate entry into chemical dependency treatment. Role playing is used to give the new team a "dress rehearsal" to practice delivering their self-written scripts one last time. Coaching is used and feedback is given to alter performances that might trigger defensive responses. If

certain members of the team cannot overcome personal anger or the desire to coddle the target person, they are asked to not attend the confrontation, but usually this is not a problem because the peer support of the team, with its singular purpose of confronting the chemically dependent person, is quite powerful in changing behavior.

Professional guidance about the most appropriate treatment for this patient will probably be sought prior to the presentation so that the urgency of the crisis can be responded to quickly and smoothly (sometimes reserving a bed at a substance abuse treatment program will prevent uncertainty and ambivalence once the patient has agreed to enter treatment).

Much care is taken to identify realistic consequences of refusal to enter treatment. Selected consequences must be something that team members control so that any ultimatum made can and will be enforced. Usually, these consequences were likely to happen anyway, if the FCCC had not been done (e.g., spouse is ready to separate, employer is ready to terminate the person's job, a child is ready to avoid visitation, a parent, friend or sibling is through bailing the CD person out of jail or financial difficulties). The physician can contribute to this by mentioning clinical potential consequences, but care should be taken in how it is said (e.g., "If you keep drinking, your cirrhosis will kill you in a year" is not a certainty, but the physician might say, "If you keep drinking, I am certain that your liver disease will progress, and I am scared that it may kill you fairly soon").

The team arranges a meeting of the entire team with the chemically dependent person at which each member, in turn, explains why he or she is very concerned about the person's chemical dependency and failure to arrest it or accept treatment for it. If the person accepts treatment, he or she is taken immediately to whatever treatment program the team decided would be best. If the presentation fails to overcome the denial, certain members of the team may inform the patient of an ultimatum, explaining what actions each person will take if treatment is not accepted or if recovery fails to occur (e.g., "if you refuse treatment now, I cannot go on living with you" or "I'm afraid that your job will be awaiting your return from alcoholism treatment but it will not be available if you return to work untreated"). These consequences most probably would have occurred spontaneously, at different times, had the patient not been confronted by the team at this time. Because the team has been formed, these actions become contingencies to reward entry into treatment and to punish refusal to accept treatment.

Usually the FCCC is effective in bringing the unmotivated chemically dependent person and the family into treatment. If it fails, it generally does so because (a) the FCCC team was not cohesive, (b) the ultimatums were unrealistic or were not enforced, (c) the treatment was poorly selected, (d) the confrontation was hostile rather than constructive, (e) the FCCC team members were ineffective in presenting their concerns because they were still exercising a lot of denial, or (f) the FCCC team did not include

CHAPTER 4: MOTIVATING CHANGE

several important and powerful enablers. If well done, the FCCC team members will generally seek treatment for themselves and recover whether or not the chemically dependent person does so. And usually the CD person finds that drinking or drug ingestion loses its charm to a large degree after such a confrontation.

It is helpful for the FCCC team to meet periodically to determine the degree of success it has had on motivating recovery. If, for instance, the patient agreed to quit drinking and go to AA meetings, the team would want to monitor that behavior and its adequacy in resolving the problems they identified. At a subsequent meeting the team might decide that the patient was not recovering because she had started substituting tranquilizers for the alcohol and was not really paying attention or being honest at the AA meetings. So they might decide to reconfront her. If the team members had made some promises to not enable her any more, the team could also give one another feedback on the enforcement of their own promises.

The family physician may develop a referral resource which can help with FCCCs or the physician may learn to perform them in the office. Such a procedure is not terribly different than what one does in delivering bad news about other terminal illnesses such as cancer. The same skills of support and firmness are needed to help the family to accept reality and to seek the appropriate clinical care for the victim and the rest of the family.

The effectiveness of social network pressure depends upon the extent to which the environmental "enablers" participate [J*Gallant, 1981; J*Liepman & Tauriainen, 1980; B*Liepman, Wolper & Vazquez, 1982]. If certain key enablers do not participate in the teamwork of planning to confront the patient about their concerns, they will probably be asked by the patient to join the opposition. For example, if John lost his job because of poor attendance and refusal to get treatment, his enabler brother might offer him a low-pressure job, but if his brother was part of the intervention team, that would not be offered unless treatment was first successfully completed.

INSTITUTIONAL COERCION

Institutional coercion occurs in industry as employee assistance programs, in law enforcement as drunk-driver programs and drug-related street crime prevention programs, and in child protection programs. In these situations, the chemically dependent person who is caught violating community rules of conduct is offered treatment as an alternative to traditional punishments (e.g., being fired from the job, losing the driver's license, going to jail, or losing custody of a child). These programs are generally very effective. However, they are vulnerable to the influence of enablers, (e.g., coworkers who make excuses for the employee's absence, lawyers who try to get the drunk-driver or street criminal off without penalty, family who offer to pretend to take custody of the child or conceal continued drug ingestion

from the child protection team. Only when all the former enablers work together as a cohesive team (with proper guidance) will the effectiveness of this strategy be optimal.

The family physician may interact with some of these programs. A patient may be referred to the physician to start on disulfiram, to be physically checked out for medical consequences, to determine whether there are psychiatric diagnoses needing attention, etc. Likewise, the patient may attempt to get the family physician involved in trying to subvert the power of these coercive programs. These programs utilize contingency contracts [B*Bigelow, Stitzer & Liebson, 1984; B*Crowley, 1984; B*Todd, 1984] to offer treatment instead of punishment, but they remove the patient's freedom to choose whether or not to continue in the program. Usually coercive treatment is justified by a serious problem with patient behavior such as driving drunk, abusing a child, committing a crime, or performing poorly at work. The patient is usually offered a choice, with treatment the more attractive of the two. If the patient decides to stop treatment, the other option is enforced. Physicians may formulate a contingency contract with a patient aside from institutional coercive programs. For example, if a patient is licensed to drive a vehicle, especially one that transports others (e.g., bus, plane), or licensed to work as a professional, the physician might ask the patient to draft a letter of self report of the addictive disorder. The letter is kept under lock and key until such time as the patient fails to return for scheduled visits or relapses. The patient has signed a request asking that it be mailed in case of these events. Until the patient relapses, the worry about its impact, if sent, provides incentive to avoid relapse.

TEACHING HINTS Supportive confrontation

Group discussion of assigned reading about the deterrents (see Table 4-1) to recovery [B*Block, 1980; J*Ewing, 1978; J*Goldwater, 1976; B*Johnson, 1973; J*Koppel, Stimmier & Perone, 1980; J*Langsley, 1978; B*Liepman, 1984; B*Liepman, Wolper & Vazquez, 1982; J*Wallace, 1977; B*Whitfield, Liepman & Williams, in press] can be followed by exploration of strategies and tactics to overcome these resistances. Case presentations of a problem patient, or of a friend or family member with a problem needing confrontation, or a film [A*Drink, drank, drunk; A*Family secret; A*If you loved me; A*I'll quit tomorrow; A*Liepman; A*Our brother's keeper; A*Soft; A*The enabler] may trigger vigorous discussion. In instances when a student actually has a serious, unresolved personal or family problem of chemical dependence, this may need to be dealt with on an individual level (see Chapter 9), but if the student is comfortable discussing the issue in a group setting (usually after it is resolved), it can be very enlightening to the group. Clinical cases also can be used in this setting.

Patient simulations, skill-building role-playing exercises, and expert demonstration of overcoming patient and family denial

and resistance can be offered by the instructor, groups of students, patients or recovering chemically dependent volunteer actors (see Chapter 3, page 8-2). Live or video observation, supervision, and coaching feedback of actual patient-trainee confrontation efforts could be arranged to measure the ability to employ selectively a diversity of tactics. Encourage coaching from the other students, from faculty, and even the patient or actor. Review or discuss criteria for success or failure of confrontations. Have one student obtain a data base, then have the other try to use that data base to confront the patient or actor. Observe successful confrontations done by faculty members. Supervise patient-trainee interactions that offer successful outcomes. Observe the trainee's reaction to apparent failure and the efforts to make plans to deal with it. Present a videotape of such a case or simulation, followed by a group discussion about the feelings evolved by such a case [B*Salint, 1987].

Discuss positive and negative social support. Assign reading about the patient's and the enabler's contributions to denial and those that may thwart successful confrontation. Conversely, consider all those measures that may support confrontation. Observe patient-trainee interactions, giving attention to the degree to which the trainee mobilizes potential support systems that may facilitate a successful outcome or confrontation.

After reading [J*Smilkstein, 1980] a case could then be made up, or one of the families in the clinic presented, for the class to determine the most appropriate time to confront. Or group interview a volunteer recovering family to determine when they were most prone to accepting help.

Have the students visit a coercive program for substance abusers and have them talk with staff and clients. Upon return, have them discuss the attitudes towards recovery expressed by new versus old clients in the programs. How do staff evaluate the motivation of the clients to recover versus to avoid the unpleasant alternatives? What is the rate of success of these programs? Have them read [J*Luks, 1982] and then discuss the ethical dilemmas of coercive treatment versus coercive punishment and versus no treatment. Have them debate the issue with some students representing the victims of drunk driving accidents, child abuse, street crime or poor work performance (e.g., errors in an airplane factory due to drugged workers), while others represent the interests of the persons sick with chemical dependencies. Try to resolve through compromise the conflicting rights and desires of the different parties.

EVALUATION: Supportive confrontation

Barriers to confrontation can be evaluated by having the resident tally the number of patients with alcohol or other drug problems in the clinical practice, and the proportion of these who are in recovery. Residents who report very few chemically dependent patients may be having recognition difficulties (see Chapter 3). Those who report a reasonable number with a small

proportion in recovery may be inhibited by barriers to confrontation. The resident may be asked to discover why he or she has so few patients or families in treatment, and some of the patients and their family members may also be asked why they have sought no treatment. The resident may be asked to tally the number of times each of the deterrents to recovery (see Table 4-1) were encountered for each of the identified families. Then, if certain deterrents stand out as more difficult for the resident, a corrective training program can be designed to help the resident to overcome these difficult deterrents. Chart review may be used to determine whether the deterrents are being listed or identified as clinical problems needing resolution. Recurrent review of the caseload of chemically dependent families over the three years of residency may offer an opportunity to also examine the stages of recovery and the rapidity of progress the families are making. The same deterrents may block such progress, so confrontational skills may be evaluated by examining stagnating cases.

Role-playing is also quite good for evaluation of confrontational skills. A patient simulation of the various deterrents to recovery could provide an opportunity to evaluate areas of strength and weakness of a resident.

CHAPTER 5: DETOXIFICATION

By Michael R. Liepman & Charles L. Whitfield

OVERALL OBJECTIVES

The teacher should train the residents and students to recognize symptoms and signs of alcohol or other drug withdrawal, and to anticipate the complications of untreated withdrawal from each specific drug class. Furthermore, they should be taught to render appropriate treatment in a timely and respectful fashion.

LEARNING OBJECTIVES: Detoxification

The student should be able to list and describe the major and minor manifestations of withdrawal from each of the major drug classes. They should also be able to describe the differences in withdrawal manifestations between different drugs within the same class. They should also be familiar with the differential diagnosis of delirium and fluent in evaluation of these other causes of delirium.

The student should be familiar with the helpful and harmful elements of social setting (non-drug) detox and medically assisted detox, and the advantages and disadvantages of each. The student should know how to select the best treatment modality for each withdrawing patient, including which local facility would be most appropriate. The student should also know some common indications for notifying a physician during social setting or self-detox.

COMMENTS: Introduction

Upon entry into hospitals, approximately a third of patients experience sudden cessation of their usual psychoactive drug intake (including alcohol, tobacco, marijuana, over-the-counter drugs, prescribed medications, and sometimes street drugs or drugs used other than as prescribed). In the ambulatory setting, patients also may suddenly cease to take their drugs for a variety of reasons such as illness, exhausted supplies, desire to quit, family pressure, etc. Patients who have developed physical dependence to these substances will experience a physiological withdrawal reaction which may cause physical harm, discomfort and concern to others (such as family, friends, nurses in the hospital). If the person has other illnesses, the withdrawal reaction could complicate the clinical course of these. While many patients treat their own withdrawal (either by tapering their own drugs slowly or by undergoing the withdrawal "cold turkey" with assistance from friends or family), some will require or request medical assistance. The goal of medically supported detoxification is to provide appropriate medical and emotional support to the patient (and family) during the withdrawal

CHAPTER 5: DETOXIFICATION

period to prevent or diminish the severity of the withdrawal syndrome and its hazardous potential impact on the patient's health and clinical course.

Problems which complicate the process can make detoxification tricky. There is danger of iatrogenic overdose during medically supported detoxification, so initial dosage selection is important [J*Jacob & Sellers, 1977].

1. The patient's ingestion pattern prior to admission is not always known (e.g., poor historian, unconscious or confused patient, denial, confabulation or lying).
2. The degree of patient tolerance (tachyphylaxis) to the drugs is often unknown, and it may vary from time to time with the same patient.
3. Concurrent illness and withdrawal both contribute to physiologic stress.
4. In hospitals, staff unfamiliarity with detox procedures or philosophy may lead to clumsy detoxification.
5. Hostile staff or family attitudes towards the chemically dependent person may interfere with optimal comfort and stability of the environment and may cause clinical mistakes in the clinical setting.

Each of these will be discussed below:

INGESTION HISTORY

Many substance abusing patients are reluctant to honestly describe their dosage patterns to authority figures such as physicians because they are afraid or embarrassed. Sometimes they may even be afraid to admit to themselves (and their family) exactly how much drug they have been taking. If the patient is not confused, delirious, or demented, a non-judgmental attitude expressed towards the pharmacological history may help bring out the truth. Patients are usually ignorant about drug kinetics and interactions, hence they fail to realize the importance to the physician of knowing about drugs of other classes that were taken recently. Some patients will not even realize that some chemicals are drugs (e.g., OTC drugs, chemicals in foods like alcohol and caffeine, or tobacco). Consequently, they may underestimate their drug intake or omit mentioning other drugs being taken simultaneously. On the other hand, some drug abusing patients may fear withdrawal or may be seeking a source of drugs to take or sell, hence they may overestimate their dosage or fabricate drugs they are not taking. It is sometimes helpful to examine pill bottles or other medications which are supplied to the doctor by the patient and family. It is also important to examine the patient for clinical signs of drug effect or withdrawal such as pupillary response, mental status, vital signs, "gooseflesh", etc. Obviously, it does not hurt to have laboratory confirmation of the presence of drugs in body fluids (qualitative urine toxicology screen or breathalyzer reading) to verify or refute the history given. Unfortunately, it is difficult to obtain rapid drug abuse toxicology screens except in emergency situations, so this may

offer less help acutely than it does for later efforts to motivate the patient to seek rehabilitation. However, if long-acting drugs are detected on the screen, it may help anticipate a protracted late onset withdrawal reaction, and it may explain why the acute withdrawal is atypical in nature. If there are several drugs in a person's body, they may interact to mask certain aspects of the withdrawal (e.g., when the opioid addict withdraws with sedatives on board, there may be less anxiety and concern about the subjective discomfort).

TOLERANCE

Tolerance (and cross-tolerance) develops in proportion to the maximum dosage that is commonly ingested [J*Kalant, 1973; B*Sellers & Kalant, 1982]. Figure 5-1 illustrates the relationship of behavioral impairment with blood alcohol levels (see also Table 1-2) in two people, one who has no tolerance and another who usually keeps the BAL at about 350-450 mg/dl. This elevation of brain tolerance, not to mention the induction of liver enzymes (liver tolerance), may account for the high dose sedative requirement of the patient during withdrawal. Competitive inhibition in the liver will initially cause a paradoxical low tolerance while the alcohol is still present in the body.

BEHAVIORAL MANIFESTATIONS

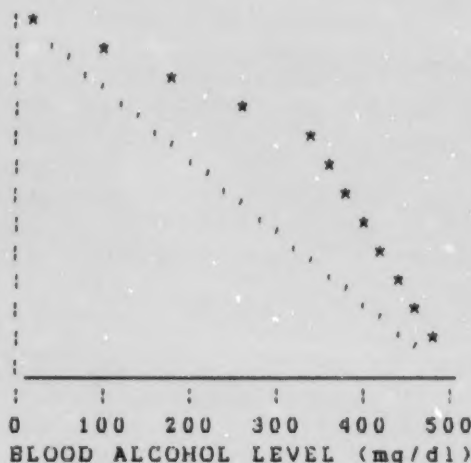
Sober
Mellow
Ataxic, slurred speech
 mild
 moderate
 severe
Lethargy
Stupor
Coma

Respiratory arrest
Death

KEY:

, = non-tolerant person
* = tolerant person

Figure 5-1: Effect of alcohol tolerance on margin of safety



CONCURRENT ILLNESS

Severity of withdrawal reaction seems to be related to the degree of physiological and psychological stress. Patients with serious life-threatening illnesses are at greater risk of serious

CHAPTER 5: DETOXIFICATION

life-threatening withdrawal reactions. For instance, a person who just suffered a myocardial infarction may not withstand the elevated pulse and blood pressure characteristic of sedative withdrawal, an epileptic may have a seizure. A person with pancreatitis, aspiration pneumonia, jaundice, esophageal bleeding, or hip fracture are much more likely to have a complicated withdrawal than a person with gastritis, a mild psoriatic flare, or a broken thumb. In some cases, as in the myocardial infarction, we understand the pathophysiology of the complicating factors, but in others all one can say is that the body is under stress. The patient should be assessed for concurrent chronic illnesses that may flare during the withdrawal. Anxiety disorders are very common in sedative and opioid abusers, hence the patient's anxiety may rise to panic proportions if incompetence or anxiety is sensed in the treating staff.

CARETAKER ATTITUDES

Attitudes of staff which subject patients to subtle or overt scorn will interfere with the working relationship. Punishing approaches like under-sedating an alcoholic patient to "make him experience withdrawal so he will remember it" or over-sedating the patient to "snow him" in an effort to reduce work load on nurses may unnecessarily kill or injure a patient during detoxification. In addition, patient and family will get the message, and this is likely to harm the patient's and family's confidence in the health care team.

DETOXIFICATION ERRORS

Staff unfamiliarity with the principles and procedures of medically supported detoxification may cause errors. Some common errors include:

1. Giving too little drug during drug substitution chemotherapy;
2. Giving too much drug during drug substitution chemotherapy;
3. Not monitoring the clinical signs of withdrawal;
4. Giving vague or confusing detoxification orders to nursing staff;
5. Not utilizing available data to verify the quality and quantity of drugs that have recently been consumed;
6. Using rigid "cookbook" schemes for detoxification which do not consider the clinical status of the patient over time [J*Jacob & Sellers, 1977];
7. Tapering the substituted drug too quickly or too slowly, or beginning the taper too early (e.g., while the patient still has cardiac arrhythmias) or too late (e.g., the day before planned discharge);
8. Using several drugs from the same class, simultaneously or serially, for drug substitution chemotherapy, hence creating a confusing situation when it comes time to taper them;

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9. Administering drugs by an erratic route (such as intramuscular librium, oral drugs in a patient who is vomiting or on nasogastric suction, or rectal paraldehyde);
10. Confusing the signs of intoxication or withdrawal with signs of other illnesses such as the stupor of hepatic encephalopathy or the anxiety from a phobic disorder.

There is a substantial risk of overdosing (hence harming) the patient who is being given unusually high doses of any drug. Patients with high tolerance (as is usually the case during detoxification) will have a lower margin of safety because of their tolerance, hence it is very important to monitor clinical condition carefully and regularly. Drug interactions may alter the detoxification (e.g., smoking cigarettes may increase the rate of removal of sedatives from the body and will temporarily elevate the pulse by 10-20 beats per minute; drinking caffeinated beverages may increase the severity of sedative withdrawal; prescribing several drugs with different catabolic kinetics may cause unpredictable or complex changes in the patient's status over time; use of pentazocine, an opioid mixed agonist/antagonist may precipitate opioid withdrawal in someone on methadone).

Concurrent illnesses may also interfere with simple detoxification. Patients with chronic obstructive pulmonary disease (COPD) who require oxygen supplementation will be in danger of respiratory arrest if breathing has been driven solely by hypoxic drive and consciousness is depressed during detoxification. Patients with liver failure may not be able to catabolize the drugs as well, and ascitic fluid may become a huge reservoir of drug which will alter states of consciousness as the quantity of ascites changes.

WITHDRAWAL SYNDROMES

Each of the drug classes have different withdrawal syndromes. The duration of action of a drug determines the time course of its withdrawal syndrome, the shorter-acting drugs having a more rapid onset and offset of withdrawal than the longer-acting drugs. Only the sedative drugs have potentially fatal complications during withdrawal (with the one exception that stimulant withdrawal mood depression could lead to a fatal suicide), and these are seen in only a small fraction of all cases (probably less than 5% in a series of late stage alcoholics). The withdrawal syndromes will not be described in detail here. They are well-described in Chapter 1 of this monograph and in a variety of references [B*Cox et al., 1983; B*Cross, Lewis & Hastey, 1974; B*Schuckit, 1979; J*Sellers & Kalant, 1976; B*Sellers & Kalant, 1982; J*Victor, 1966]. The description of the withdrawal syndrome must be kept in mind while reading the subsequent sections of this chapter.

CHAPTER 5: DETOXIFICATION

SUBSTITUTION CHEMOTHERAPY

The basic strategy of medically assisted detoxification is to reduce the rate at which the level of drug effect disappears from the body, and hence to slow the body's readjustment to life without the drug. Alcohol, heroin and morphine, triazolam, inhalant anesthetics, cocaine, amobarbital are all examples of very short-acting drugs whose withdrawal is rather acute. By either substituting a longer-acting drug or by gradually tapering the dose of the original drug, the severity of the withdrawal reaction can be minimized. Consequently, the strategy for medically assisted detoxification has five phases:

1. Pre-detoxification
2. Stabilization phase
3. Maintenance phase
4. Taper phase
5. Drug-free phase

Prior to cessation of substance use is called pre-detoxification phase. During this phase, the patient is controlling intake without physician input or control. During the initial days after cessation of ad lib substance ingestion (stabilization phase), the physician clinically titrates dosage of the selected substitute drug to approximate the pre-cessation body drug level. This is done empirically, based on patient need and response to medication. Once stabilized, the dosage is titrated to keep body level constant (maintenance phase) until the appropriate time to begin the taper. During the taper phase, the dosage is adjusted to reduce the body level gradually. Once the taper is completed, the patient enters the drug-free phase. Figure 5-2 graphically illustrates these phases. Clinical judgment must be used throughout the process to ensure that the drug dosage is correct based on clinical signs of withdrawal and intoxication. Wide variations from one patient to another makes rigid "cookbook" schemes (which residents tend to like for their simplicity), dangerously unreliable. Fortunately, patient comfort and confidence in the staff helps reduce the need for medications, but both emotional and physiological stress exacerbates sedative and opioid withdrawal. The clinical decision as to when it is best to make a transition between one phase and another requires knowledge of the patient's condition and situation. Stabilization is best done in a setting where the patient is not controlling drug intake alone, though patient input is very important in this phase. Maintenance phase can be very helpful in delaying taper until the patient's clinical condition is otherwise stable (e.g., until the myocardial infarction patient can withstand tachycardia).

FAMILY MEDICINE CURRICULUM GUIDE TO SUBSTANCE ABUSE

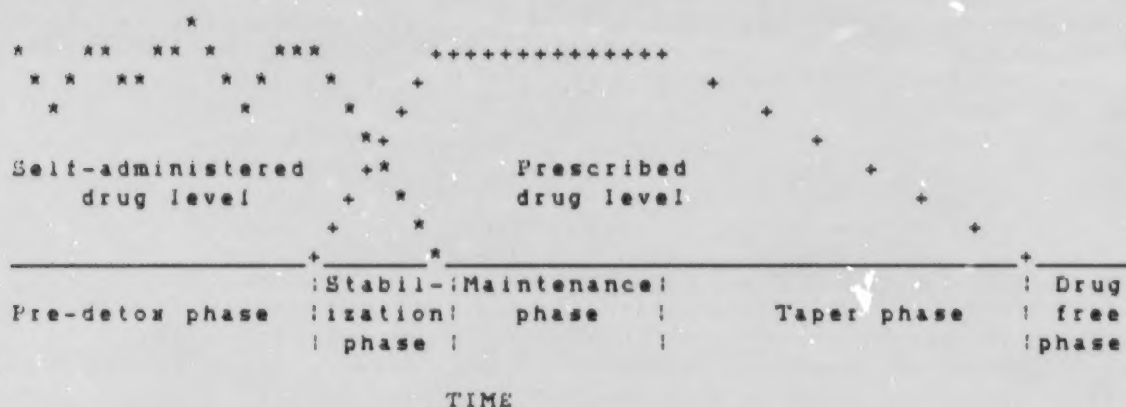


Figure 5-2: Body drug levels during phases of medically assisted detoxification using substitution chemotherapy

Calculating Dosages

In order to determine how much drug to give a patient during the stabilization phase, one may use the history as a guide, then empirically administer drug based on clinical need and response. For instance, a sedative detox patient could be given a sedative dose hourly until lethargic whenever resting pulse rate exceeded 110. Provided the patient was given a large enough dose of sedative to bring the withdrawal signs under control in the first day, the total of all doses administered during the first 24 hours would reveal the patient's need. If the dose was too large, lethargy and normal pulse would cause staff to halt administration of sedation. If the dose was too small, the patient's condition would deteriorate progressively until the hourly dose was increased. During the second day, additional doses will probably be needed. This can again be administered empirically. Once the patient's clinical condition has stabilized, this terminates stabilization phase of detox.

During the subsequent days, known as maintenance phase, the goal is to maintain the clinical stability of the patient. During this phase, it is necessary to tally the daily dosage received initially in order to compute the amount of accumulated drug remaining in the body. One first determines how many half-lives fit into 24 hours. Then one takes 2 to the power of the number of half-lives and divides that number into the prior day's dosage as follows:

$$R_n = D_{n-1} / (2)^H$$

where n represents the day, R is the amount of drug remaining in

the body on this day, D is dosage on this day, and H is the number of drug half-lives that fit into one day. For example, if the drug has a 12 hour half-life, then $H=2$, or if the drug has an 8 hour half-life, then $H=3$. The value of R is computed daily and added to the dosage administered that day to find the daily body dosage thus:

$$\begin{aligned} \text{DBD}_n &= R_n + D_n \\ &= R_{n-1} + D_{n-1} / (2)^H + D_n \end{aligned}$$

where DBD is the daily body dosage on a particular day. For an illustration of this computation, see Table 5-1. Note that the half-life may be found in the PDR or other reference texts, but that liver or renal disease or other drug administration may alter the half-life to some degree. (Table 1-3 lists half-lives for benzodiazepines.) In order to maintain the patient in a clinically stable state, the DBD should be kept constant by administering just enough drug each day to replace catabolized drug. This is easily calculated using the formula:

$$D_n = \text{DBD}_n - R_n$$

However, clinical judgment will still be necessary because the formula assumes that the catabolic rate does not change, but it might if other medications are added or removed, or if the liver or kidneys lose or regain function.

Once the patient is ready for the taper phase, the DBD is gradually lowered until the patient is drug free. Probably the most rapid rate of DBD reduction that patients will tolerate well is a taper over five days (roughly 20% of maintenance phase DBD per day). After a protracted maintenance phase in outpatient methadone maintenance programs, the taper of methadone DBD may be done usually over no less than 21 days and is often done over many months, since failure to tolerate the reduction may lead to relapse of illicit heroin abuse, return to crime, etc. For inpatients who are anxious to be discharged and who do not have a ready source for relapse (while in the hospital), the 5 day detox may be acceptable. But beware of precipitating a discharge against medical advice to obtain drugs by prescribing too rapid a taper. Also beware of discharging the patient too soon after becoming drug free; the craving is still present in a quite strong form for days [J*Mathew, Claghorn & Largen, 1979]. A single-dose diazepam treatment has been suggested for alcohol withdrawal which involves giving the patient a loading dose of diazepam on the day of admission [B*Sellers & Kalant, 1982]. Since diazepam catabolism in most people is roughly 20% per day, the taper is performed automatically by the body without requiring any additional drug. This approach is inappropriate for complicated detox cases who may require a maintenance phase prior to taper.

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Table 5-1: Calculation of daily body dosage for chlordiazepoxide^a

DAY ^b	1	2	3	4	5	6	7	8	9	10
Dose given today	250	650	400	400	250	150	75	25	0	0
Remainder from yesterday	0	125	387	393	396	323	236	155	90	45
Daily body dosage level	250	775	787	793	646	473	311	180	90	45

^a Assuming half-life of 24 hours

^b Note that days 1-2 in this example are stabilization phase, days 3-4 are maintenance phase, and days 5-10 are taper phase.

Drug Selection

When selecting the drug to use in substitution chemotherapy, one usually chooses a drug that has cross-tolerance to the one used by the patient. In some cases the same drug is used. Many alcoholics will detox themselves by tapering their alcohol, when they are able to exert enough control over intake to complete the task. In the clinical setting, choice of drug might be governed by issues such as (1) availability of drug in hospital formulary; (2) familiarity and acceptability of drug to physician and nurses; (3) half-life of drug is convenient for physician schedule (longer-acting agents tend to need less watching in all but emphysematous patients); (4) route of administration is appropriate to patient's condition; or (5) catabolic mechanism (renal excretion in uremic patient or liver breakdown in jaundiced patient may be problematic unless the patient is carefully monitored clinically) [J*Kaim, Klett & Rothfeld, 1969]. One must always keep in mind that the patient will have cross-tolerance, hence probably will require large doses in comparison to those used with non-tolerant individuals. Hence, cost of the medication may be a consideration, too.

CLINICAL APPLICATIONS

For alcohol or other sedative withdrawal by substitution chemotherapy, the procedure begins with the stabilization phase. Like treatment of diabetic ketoacidosis, the clinical signs of withdrawal dictate the administration of sedative doses. Resting pulse >100, fine tremors of the digits, agitation or pacing and night-time insomnia are good clinical signs of alcohol/sedative withdrawal provided they are not attributable to other concurrent disease processes or other factors (e.g., the heart rate is up

due to acute blood loss; the insomnia is due to a noisy room). Vital signs should be monitored at least every 1-2 hours while the patient is awake (if the patient is asleep, obviously enough sedation has been given). Beware of patient intake of caffeine or nicotine. The former may counteract the sedative effect, while the latter may transiently (i.e., for 10-15 minutes) elevate pulse and blood pressure. An initial test dose of 50-100 mg of chlorthalidone, or the equivalent dosage of another drug will determine whether the patient has a high or low tolerance. Then these doses can be repeated hourly as needed to control the pulse, tremor, agitation or insomnia. At bed-time, a larger dose may be given to eliminate the need for frequent awakenings, once the patient's tolerance has been adequately established. By this scheme, the most a patient could receive in the first 24 hours is 0.6-2.4 gms of chlorthalidone, which should suffice for any case. Most patients would receive less than 600 mg. Patients who tend to require sedative treatment include (1) patients who are hospitalized in intensive care areas; (2) patients who are hospitalized with acute medical problems; (3) patients under the care of treatment staff who are not trained for nondrug detoxification; and (4) patients for whom nondrug detoxification has failed. Some patients may not require much or any sedation during detoxification. These patients are usually (1) relatively healthy, (2) young, or (3) have not been drinking heavily recently (such as a person who just relapsed for a few days prior to admission).

Once stabilized, the sedative dependent patient may then be maintained on dosage sufficient to keep DED constant until time for taper. At this time, the dosage is no longer given from a sliding-scale order, rather it is given in divided doses throughout the day. Care should be taken to consider the activities of the patient and the side-effects of the substitution chemotherapy drug. It is often advisable to give 40% of the daily dosage at bedtime to take advantage of the soporific effect at night and to avoid it during the day. Indefinite sedative maintenance is not recommended for patients because (1) there is a significant risk of dependence, (2) tolerance will develop such that gradual increases in dosage will be required over time, and (3) side-effects from sedatives may cause problems (e.g., memory problems, mood depression, impaired judgment, impulsive behavior, lack of appropriate concern about life problems).

Once taper phase begins, the DED is reduced usually by 10-20% per day by reducing daily dosage (10% per week for outpatients). As doses get smaller, it may be impossible to obtain the calculated doses as suggested by the formula. One strategy is to give the total daily dose at bedtime. As the DED falls further, there may be several days during which no new drug is administered.

If the patient develops delirium, 3-6 days after cessation of drinking or longer after withdrawal from longer-acting sedatives, it may be a withdrawal delirium. However, the patient should also be evaluated for possible other causes of delirium such as:

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1. electrolyte imbalance,
2. post-ictal state,
3. vascular compromise,
4. meningitis,
5. intracerebral pressure syndrome,
6. hepatic encephalopathy,
7. Wernicke's disease,
8. drug toxicity (e.g., bromides, corticosteroids, stimulants, atropine, and hallucinogens), interaction or withdrawal,
9. uremia,
10. hypoxia,
11. hypoglycemia,
12. high fever,
13. thyrotoxicosis,
14. hypoparathyroidism,
15. congestive heart failure,
16. acute pancreatitis,
17. postanesthesia confusional states.

The specific manifestations of delirium tremens are extreme hyperactivity of (1) the psychomotor nervous system, evidenced by extreme agitation, restlessness, and usually hallucinosis, misperception of stimuli and mental disorientation; and (2) the autonomic nervous system, as evidenced by sweating, tachycardia (120-130 beats per minute), dilated pupils, and low-grade fever. Haloperidol (Haldol[R]) may be given (e.g., 5 mg) once or twice daily. If the sedation has been sufficient to keep the adrenergic signs of earlier withdrawal under reasonable control (e.g., pulse < 90 and patient is able to sleep), further increase in sedation is not indicated. Physical restraints may be necessary to protect the patient from injury, however interpersonal conversation to help orient the patient to the surroundings with staff, a friend, or a family member may suffice and is certainly less demeaning. Increasing sedation at this time may reduce risk of harm if the patient is "snowed" to the point of remaining in a stupor much of the time, however the patient may become a management problem at intervals between stupors, and the patient will not be able to assist staff in handling basic bodily needs such as urination, defecation, eating and drinking. Furthermore, if the delirium is caused by another etiologic factor, assessment will be more difficult in a stuporous patient.

Two other types of drugs have been used in the past for alcohol detoxification in addition to sedatives: antihistamines and phenothiazines. There is little rationale for the use of phenothiazines today because of their potential side effects, and the much wider margin of safety provided by other sedatives, particularly by the benzodiazepines. The antihistamines are still used for sleep, particularly at the end of the taper phase for insomnia, but caution should be exercised to avoid fostering a new drug dependence.

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Other Hypnosedative Withdrawal

The detox for other sedatives is essentially the same as for alcohol, with the exception that the time course is slower for the onset and offset of the withdrawal signs and symptoms. This is a direct consequence of the catabolic kinetics of the drug in question.

Opioid withdrawal

A similar scheme may be used for opioid dependent patients using a long-acting drug such as percodan or methadone. For a person with tolerance, daily doses of methadone as high as 40-120 mg have been reported in methadone maintenance programs. Usually it is safe to use a 10 mg test dose, followed by 10 mg doses every few hours until the patient reports relief of withdrawal symptoms or pain. Once the patient is stabilized, the maintenance phase may begin.

Methadone maintenance is widely accepted as a method to prevent opioid withdrawal discomfort and to block the euphorogenic effect of street heroin (by saturating the opiate receptors). While the former is true, the latter may be open to question in some patients. Many patients are maintained on methadone for many years (during which they are supposed to be getting some counseling).

As mentioned earlier, the taper may go as quickly as 21 days or as slowly as months to years. Some programs do a "blind" detox where the patient is not told when and how much the dose will be reduced. One problems with methadone detox schemes include patient ingestion of other drugs during the detox which may potentiate the remaining body methadone.

Stimulant withdrawal

Withdrawal from the psychomotor stimulants is described in Chapter 1. Usually clinical detox does not require drug substitution. One exception to this may be the detox from smoking cigarettes. Patients may detox themselves by lengthening the interval between cigarettes or reducing the length of butt they leave unsmoked. In addition, there is now available a new nicotine chewing gum (Nicorette[R]) which can be dispensed with a prescription.

OTHER WITHDRAWAL APPROACHES

There has been recent attention to several alternative approaches to detox that are fundamentally different from the substitution chemotherapy approach described above.

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Social setting detoxification

Ambulatory alcoholics with uncomplicated withdrawal may successfully complete detox without medical complications in a cost-effective relatively comfortable home-like non-hospital setting. Usually staffed by recovering alcoholics but without physician coverage (except emergency room back-up), the strains of soft music replace the hospital overhead paging system, chicken soup and fresh apple pie substitutes for the bland hospital diet, and caring staff who "have been there" chat respectfully with the patients, providing reassurance and reality orientation. Patients talk with and support one another. The environment should be quiet, partially lighted, and should always have familiar people available to meet the patients' needs. Features like a low bed, comfortable furniture, regular clothes, and a predictable schedule will help improve the patient's clinical condition. Likewise, optimism about short- and long-term recovery and coping with all of the patient's life problems is helpful, being careful to avoid focussing too much attention on them now (the AA principle: "Take life one day at a time" often comes in very handy at a time like this). Some countertherapeutic environmental conditions are (1) room lights off or very bright; (2) marked shadows; (3) loud or abrupt noise; (4) high bed; (5) IVs and tubes; (6) staff hostility, even if subtle; (7) negativity or pessimism; and (8) physical restraints.

Hospitals, by their very nature, take away some degree of personal control over one's life. While this is often necessary for the safe and efficient operation of a hospital, it may be harmful to the confused withdrawing alcoholic or drug dependent patient. Hospitals usually are characterized by bright lights, lots of noisy activity and emergencies announced on the overhead paging system or beepers, overworked staff who cannot readily be available to the alcoholic patient at the drop of a hat. To a patient whose nervous system is going through a period of hyper-reactivity to stress, the hospital may be the worst place to be, if the patient is otherwise healthy. This modality has demonstrated its overall safety for otherwise relatively healthy alcoholics and its cost-effectiveness compared to hospital care [J*Whitfield et al, 1978]. However, it must be emphasized that these programs do selectively get referrals of primarily patients who need less medically sophisticated care. While social setting principles can be put to use in hospital and home detoxification approaches, the withholding of sedative medications from hospitalized withdrawing alcoholics cannot be justified by the social setting detox success.

Patient selection is the key to proper utilization of non-drug social setting detox facilities. Patients with a history of previous withdrawal seizures, if not treated with benzodiazepines, should receive phenytoin for five to seven days [J*Sampliner & Iber, 1974]. Non-drug facilities usually cannot dispense phenytoin, although occasionally a program will let a patient take medication such as this if prescribed by an outside doctor. Seizure risk would be an indication for hospital detox. Most alcoholics, unless their withdrawal period was proceeded by

CHAPTER 5: DETOXIFICATION

protracted nausea and vomiting, or severe diarrhea, are over-hydrated and do not require intravenous fluids. If the patient can take oral nourishment, juice, soup and gelatin can be provided in ample quantities. Only in cases of severe dehydration will fluid intake require hospital care. All patients in acute withdrawal should have thiamine administered immediately. Parenteral route is better than oral. This can be given in the emergency room or doctor's office prior to admission to the social detox center. If a patient is clinically unstable or if the patient has an acute or chronic medical illness that requires medical monitoring or medications, probably the patient is suitable only for a medical detox facility (e.g., hematemesis, fever greater than 100.5 degrees F, dyspnea or tachypnea >30, persistent tachycardia >120, chest pain, seizure, shaking chills, recent head injury, and delirium tremens). Likewise, if a patient exhibits psychotic thinking or difficulty controlling impulses (e.g., violence, suicide), then the patient would be more appropriate for a psychiatric setting detox.

Several advantages to nondrug detoxification are (1) its shorter duration; (2) its lesser expense; (3) its administration by nonmedical personnel; (4) the earlier diagnosis of psychiatric conditions it allows; (5) the lower risk of becoming cross-addicted to another drug; (6) the patient's alertness and ability to participate actively in treatment; (7) the patient will be able to remember the experience; and (8) the patient is more likely to be referred to an alcoholism or drug rehabilitation program at discharge.

Symptomatic drug detox

The Haight-Ashbury Free Medical Clinic has been offering a non-opioid ambulatory detox for opioid addicted patients [B*Newmeyer & Whitehead, 1978] in which patients are treated for symptoms of their withdrawal with non-narcotic drugs. For example, the gastrointestinal symptoms would be treated with an antiemetic like prochlorperazine (Compazine[R]) and insomnia might be treated with a sedating antihistamine. The retention rate of patients in this modality is low, but a significant number of cases do get successfully detoxed from opioids and other drugs in this way.

Acupuncture assisted withdrawal

Acupuncture probably stimulates some hormonal pain relieving factor which arises from the pituitary and can be blocked by naloxone [B*Goldstein, 1978]. Clinical utilization of acupuncture in opioid withdrawal has been reported to be moderately successful [J*Kao & Lu, 1974; J*Wen & Cheung, 1973], though hard to market (free) to western addicts [B*Newmeyer & Whitehead, 1978]. Acupuncture has also been tried with tobacco smokers with good success [B*Rutledge, 1983].

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Family home detox

One approach to minimize the recidivism of drug detoxes for young heroin addicts utilized the parents as detox technicians in the home [J*Stanton et al., 1978]. The family members were taught how to do it properly and were involved in the recovery through their initial involvement in the detox.

Clonidine detox for opioids

Alpha-adrenergic blockers such as clonidine have been tried in opioid withdrawal detoxification [J*DiGregorio & Bukovinsky, 1981; J*Gold, 1980; J*Gold, Redmond & Kleber, 1979; J*Riordan & Kleber, 1980; J*Washton, Resnick & Rawson, 1980]. The use of clonidine as a non-narcotic aid to opioid withdrawal has been quite successful, though not without hazard due to hypotensive events. Clonidine has found its ways onto the streets for unsupervised use by addicts.

Propranolol for alcohol detox

Beta-adrenergic blockers have been used successfully to block alcohol withdrawal induced arrhythmias [J*Zilm, et al., 1980]. There is also some improvement of the tremor and the subjective anxiety with propranolol.

Low dose alcohol detox

It has been suggested that ethanol is an essential metabolic ingredient, and its sudden absence leads to a metabolic deficiency which is posited to be the etiology of the withdrawal syndrome [J*Gower & Kersten, 1980; B*Schmitz, 1978]. The same authors advocate low dose (i.e., 30 cc./hour) ethanol ingestion or iv drips to ward off the withdrawal syndrome. They claim anecdotal success, but have not demonstrated comparative efficacy and safety in controlled experiments. Given the high rate of success with the social setting approach, this may have little to do with the homeopathic ethanol treatment. Nevertheless, there are numerous surgeons who use this technique, apparently in response to the above cited article.

TEACHING HINTS: Detoxification

In the classroom, offer lectures and discussion on withdrawal and medically assisted and social setting detoxification. Have the students each read a different article about detox [B*Baum & Iber, 1980; J*Benzer & Cushman, 1980; J*Coates, 1927; J*Coleman & Evans, 1975; J*DiGregorio & Bukovinsky, 1981; J*Gold, Redmond & Kleber, 1979; B*Gross, Lewis & Hastey, 1974; J*Jacob & Sellers, 1977; J*Kaim, Klett & Rothfeld, 1969; J*Resnick et al., 1977; J*Sampliner & Iber, 1974; B*Schuckit, 1979; B*Seixas, 1971; B*Sellers & Kalant, 1982; J*Sellers & Kalant, 1976; J*Stanton

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et al., 1978; J*Thompson, 1978; J*Tornabene, 1974; J*Victor, 1966; J*Washton, Resnick & Rawson, 1980; E*Whitfield, in press; J*Woo & Greenblatt, 1979] and have them present what they learned to one another. Try to have a physician who is very skilled in detoxification on hand to moderate the discussion. If possible, devise several case presentations of progressively more complex difficulty to present to the students to challenge them to utilize their new knowledge in clinical problem-solving.

In the outpatient setting, have the resident attempt an outpatient detox with a patient who seems appropriate for this.

In an inpatient setting, assign the resident a patient who is being treated for withdrawal and have the resident observe the patient and write detoxification orders under supervision. Have the residents find a case of delirium tremens in the hospital to show to the whole group, preferably with the guidance of an experienced faculty to point out the salient features.

Arrange for site visits to local medical and social detoxification facilities. Have them inquire about the type of patients they serve, who they send away, why and to where are they referred. After the site visits, have the students critique each of the facilities in terms of features of their milieu which might help or hinder the uneventful detoxification process. If it is possible to have the residents or students "moonlight" or take a brief (even for just a weekend or overnight once or twice) clinical rotation at one of them, this would be a very valuable learning experience.

Either in the detoxification facilities, or by inviting recovering volunteers from the local AA groups, have the students interview recovering chemically dependent patients, and have them specifically question the patients about their own experiences with withdrawal including attempts at self-detox. If family members are available, have the students question them about how it was to watch withdrawal both in a medically assisted setting and when done at home without assistance.

EVALUATION: Detoxification

Give cognitive examinations, either written or oral. Observe learner comments during discussions on tolerance, major and minor symptoms of withdrawal, and choice of drug and dose. Examine patient management problems that the students completed.

Observe the residents' methods of patient management in a clinical setting, watching for the recognition of the withdrawal syndromes, the approaches used to treat them, and the clarity and confidence in communication with nurses. Observe the patient's clinical response to the prescribed treatment and watch the resident for attentiveness to and respect for the clinical signs and symptoms.

CHAPTER 6
SUBSTANCE ABUSE REHABILITATION
By R. C. Anderson, J. V. Fisher, C. L. Whitfield & M. R. Liepman

OVERALL OBJECTIVES

The family medicine resident should learn to initiate an appropriate treatment plan, make appropriate referrals, and collaborate in long-term follow-up care with the substance abusing family. When encountering a relapsed recovering patient, the resident should be able to analyze triggers to the relapse and formulate a modification of the recovery plan to avert future relapses by this trigger. The family medicine resident should become facile in referring the patient and family to the self-help recovery groups, and working collaboratively with them.

TREATMENT PLANNING & REFERRAL

LEARNING OBJECTIVES: Treatment Planning & Referral

The student should be able to cite recovery rates of alcoholism and other addictive disorders with and without rehabilitation. The student should be able to define criteria for treatment success. The student should be able to list factors associated with high or low recovery rates. The student should be able to list methods used in substance abuse rehabilitation.

The resident should be able to plan and conduct a patient referral for substance abuse treatment, listing criteria for selecting the referral source in the local community, and giving alternatives if the first choice is not successful.

COMMENTS: Treatment Planning & Referral

Once the family physician makes a diagnosis of substance abuse, the next step is treatment planning and motivating the patient and family to begin recovery. Treatment options should be carefully selected to match family resources and needs. Then these options can be recommended to the family so they can become involved from the very beginning by making the final selection.

The selection of treatment options depends mostly upon assessment of needs, but resources must also be considered [J*Liepman, 1980]. Options for detoxification depend on the type of drug (or drug combination) used, the amount used, the duration of use, and the physical and mental condition of the patient (see Chapters 1 & 5). The degree of social support that is present to complete a detox may also be an important variable. Physical dependence is not always present in chemically dependent patients.

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and drugs such as marijuana and cocaine appear to foster few or no physical withdrawal symptoms. For example, a patient who is coming off cocaine does not need hospital care for detoxification, but an alcoholic with high intake, past history of withdrawal seizures and mild jaundice will be much sicker in the hospital. An alcoholic with no other problems could probably survive a home detox or "drying out" at a social setting facility, but a patient who has lost all self control to abstain and who has little constructive social support would be unlikely to complete a home detox.

Treatment options range in intensity from extended inpatient rehabilitation to ambulatory abstinence and involvement in a self-help recovery group. Treatment options will vary with each community, and it is incumbent upon the family physician to be familiar with the local and regional treatment and recovery aids that are available. This awareness includes knowledge of qualifying factors such as third-party coverage, catchment areas, and entrance requirements. If the family physician is not aware of these details, and does not consult someone who is, a lot of effort in motivating the patient and family to seek treatment may be wasted if they are not accepted. Such an error could also damage the family's confidence in the physician. It never hurts to phone the facility before attempting to refer a case there.

Description of the facility and its program is also a necessary task for the family physician. The family will have lots of questions about what kind of commitment they are making, and what they will get from their investment. Treatment philosophy is also important to consider. For instance, if a patient who wishes to be taught to drink in a controlled fashion is sent to an abstinence oriented rehabilitation program, it is unlikely that the patient will agree to enter the program.

The method of motivation may also be important. Assume that a person seeks treatment pending a prison sentence. The chosen rehabilitation facility requires that all legal matters be resolved before treatment begins, because court dates may interfere with attendance at therapy sessions, or the judge may pull a patient out of treatment and put him in jail in the middle of the program.

Transportation or work schedules of family members or their willingness to participate in family treatment may be important. The duration of the program may conflict with permissible sick-leave of the patient's employer.

INPATIENT TREATMENT

Inpatient care may take place in a hospital rehabilitation unit or in a long-term residential therapeutic community. Treatment philosophies and the major thrust of programs vary considerably. This results in differences in program components and emphasis. Major components that can be found in rehabilitation programs include:

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1. education about the disease model and the consequences of substance abuse.
2. psychodynamic individual oriented psychotherapy.
3. exposure to self-help recovery programs and their philosophies.
4. group therapy of a supportive or confrontive nature.
5. milieu therapy, emphasizing taking responsibility for one's actions and its social consequences.
6. occupational therapy to build self-esteem, train new hobbies and skills, and to enhance work skills and employability.
7. relaxation therapy and anxiety management (D'Caddy & Block, 1983).
8. biofeedback and pain control.
9. social skills training such as communication and assertion training.
10. family interactional therapy (see Chapter 5).
11. aversion therapy (D'Caddy & Block, 1983).
12. recreational therapy and life restructuring.
13. controlled drinking training (D'Caddy & Gotthelf, 1983; DeLoeband, 1983).

The family physician may or may not be able to participate during inpatient treatment, depending upon the location and the type of facility. But in any case, the family physician should try to remain in contact with the staff of the facility during rehabilitation to assess progress and to communicate how the rest of the family are doing, and to become a party in aftercare planning. Once discharged, the doctor may be asked to prescribe and monitor antabuse if it is to be used, and avoid the prescribing of abusable psychoactive drugs such as opioids or minor tranquilizers and sedatives.

Hospital based rehabilitation programs are usually short-term (e.g., approximately 28 days in duration) in order to accommodate third party coverage. Veterans programs may be longer because of different payment mechanisms and different needs. When a patient is about to be discharged, an aftercare treatment plan is devised to address the unresolved needs that have been discovered and to provide ongoing support during the recovery and any relapses that may occur. Referral to a half-way house at this time is helpful for those who have the need to develop a new social support system (either because they had little social support prior to entry into the program or because their natural social support system generates a high risk of relapse and they have decided to disengage from this network). Since inpatient care is a rather artificial social environment, good transitional support is crucial to keeping the relapse rate down and minimizing the severity and duration of relapse as the patient returns to the real world (usually surprised by the lack of environmental support for their sobriety).

Long-term therapeutic communities (often 6 months to 2 years or more) provide intensive rehabilitation of life skills. Such programs are usually most appropriate for persons with per-

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sonality disorders in addition to the chemical dependence who find sober life impossible to bear until they have made significant changes in behavior and attitudes.

Patients with other psychiatric disorders such as affective and psychotic disorders will need care for both the chemical dependence and the psychiatric disorders. The duration of treatment may vary according to the individual needs of the patient. Staff expertise in both areas is a necessity. Patients with medical disorders (e.g., diabetes, epilepsy) or disabilities (e.g., deafness, emphysema, paraplegia) may require special treatment settings that can accommodate their special needs.

Studies of treatment outcomes have resulted in a wide variety of recovery rates, depending upon the treatment offered, the types of patients served, and the measure of treatment outcome selected. There is evidence that alcoholism rehabilitation results in recovery in over 50% of all cases, and some studies report a recovery rate of 75% which is far higher than the spontaneous recovery rates for alcoholics or other drug abusers [B*Baekeland & Lundwall, 1977; S*Litman & Topnam, 1983; J*Tuchfeld, 1981; E*Valliant, 1983; J*Valliant, 1966]. Family involvement appears to enhance recovery rates (see Chapter 8).

OUTPATIENT REHABILITATION

Outpatient treatment has many of the same features as inpatient treatment, but it usually is of far lower intensity. It is designed to cost less in terms of money and time and is compatible with continued involvement in the social environment such as employment and family relations. Outpatient care may be intensive (four or five evenings a week for four to five weeks) or may be offered twice or three times a week for several months. Many programs use and introduce self-help groups, multiple family therapy groups, educational classes, and provide aftercare. The results of outpatient care appear to be as good as those of inpatient treatment, although the patient selection is far different.

It is important to be careful in the selection of patients for outpatient care. The patient should have a reasonable support system, should not be a danger to him- or herself or others, must be able to function in a nonstructured environment, must not have failed in outpatient treatment in the past, must be motivated, and have a low volume or duration of drinking.

Outpatient treatment often requires that the patient be detoxified before beginning treatment. The use of disulfiram (Antabuse [R]) should be considered for alcoholics until a foundation (at least 6-12 months) of abstinence has been established. For opioid dependent individuals, methadone maintenance with counseling is commonly available [E*Schuckit, 1977]. The quality and quantity of counseling is variable from program to program. One way to assess this is to inquire about the proportion of patients attending the clinic who are "drug free" versus on

methadone. Those clinics with very few "drug free" clients are probably not able to retain clients beyond the methadone detox, hence the only clients who come or stay are those who must come to avoid the methadone withdrawal syndrome. On the other hand, programs with a high proportion of "drug free" clients must be offering some good counseling to have attracted clients for counseling alone. Methadone wards off the withdrawal syndrome and decreases craving and the "high" feeling after using heroin. It can be given once daily, usually in doses between 20-80 mg., along with regular outpatient counseling. In some locations other drugs are being used on an experimental basis such as LAAM (11-alpha acetylmethadol--a long acting methadone analog given every other day) or naltrexone (a long acting naloxone analog which blocks opioid receptors). Naltrexone users cannot get "high" on opioids and must be detoxed before starting treatment. All of these patients can get "high" by using non-opioid drugs. Usually the outpatient drug programs will monitor drug (but not alcohol) abuse in clients by frequent surprise urine toxicology screens and will discipline clients who continue to abuse drugs despite being in treatment by detoxing them off methadone and discharging them from the clinic. It is paradoxical that they are "punishing" their clients for the symptoms of the very disease they are treating by throwing them out of treatment. A more rational approach is to consider advancing them to inpatient or residential care if they cannot control drug use in the ambulatory setting.

Self-help recovery groups may be sufficient for some patients to achieve and maintain sobriety. Many thousands of people have achieved sobriety in this way. The usual recommendation is to attend 90 meetings in 90 days, and to find a sponsor. But patients who are unable to achieve sobriety in this way may need additional counseling.

Outpatient services are often offered by large companies through Employee Assistance Programs (EAPs). Usually the services are limited in scope and depth to diagnostic exploration, motivation and referral. Commonly staff are limited in training, numbers and clinical supervision. It is not atypical for staff to be a recovering alcoholic counselor and a secretary for a plant of 5000 employees. Outpatient services are also offered in conjunction with the family court, drunk-driver programs, and probation offices which focus on alcohol and other drug abuse problems. Usually the scope of these services is also limited. Quite frequently, drunk-driver programs are primarily educational with diagnostic, motivational and referral components to help sort alcohol abusers from alcoholics and send the latter to more appropriate care providers. Before considering the care of these programs adequate treatment, it is important that the family physician check out the qualifications of the staff and their own perceived role in care, as well as the impression of other experts in the community about their role and skill.

Outpatient aftercare following inpatient, residential or intensive outpatient rehabilitation may continue for months to years. Key elements of aftercare include addressing the adjustments to a sober lifestyle, coping with the response of the so-

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cial environment to personal changes, including in some instances, divorce or family breakup, loss of job, or changing one's friends. Special issues may be addressed in long-term counseling such as grief over losses, guilt over past misdeeds, self-esteem and identity (sexual, marital, work, religious) and behavior in relationships, depression, or anxiety.

Outpatient services provided by an individual therapist in a private practice mode or in a mental health or social service agency may be helpful, but caution should be exercised when there is not proven expertise in substance abuse care. Since substance abuse training has not been available or required in many professional training programs, psychiatrists, psychologists, social workers and other counselors may not have the training to provide good care. Likewise, agencies might not have someone on the staff with the skill to supervise others who are learning to do substance abuse care. A rule of thumb to use in judging the competence of such programs is to check the background of staff and then talk with the therapist to determine whether this person is planning to utilize the self-help recovery programs, maintenance on disulfiram or methadone, other community resources such as detox facilities and specialized rehabilitation programs versus benzodiazepine or other abusable psychoactive drug maintenance, and individual-oriented psychotherapy. Usually individual-oriented growth psychotherapy is contraindicated in outpatient care until the patient has accumulated at least 2 years of stable sobriety, because the stress of the psychotherapeutic process often stimulates substance abuse relapse. Freud has advocated that the patient remain abstinent throughout psychotherapy, and most experts in substance abuse agree that this is a necessity. If a patient desperately needs psychotherapy to remain abstinent, an inpatient or residential setting is an appropriate place to do this. Another question to ask the therapist is how abstinence will be verified. If self-report is the only method, one might question the naivete of the therapist. If the therapist is knowledgeable, a breathalyzer (B*intoximeters), or drug abuse toxicology screen will be a regular part of the therapeutic regimen, or other methods will be used such as the sweat patch (J*Phillips, 1982, J*Phillips et al., 1984, J*Phillips & McAloon, 1980) or periodic enzyme assays or collateral reports by family informants.

TEACHING HINTS: Treatment Planning & Referral

Students and residents can watch a video (A*Resources for Alcoholism treatment) and then visit local treatment facilities to determine the types of patients they accept and the kinds of treatment they offer. Block rotations or half-day visits are possible methods for achieving this objective. Students may be admitted as patients for a weekend (R*Wittmers) to see what it is like. Residents may clinically follow their own patients which they referred to the rehabilitation facility or they may provide follow-up primary care for a patient they met during a block rotation in the facility. Participation in a therapy group would give the resident an excellent taste of the recovery process.

FAMILY MEDICINE CURRICULUM GUIDE TO SUBSTANCE ABUSE

Use films (A*Loosening the grip, A*The physician's role, and readings (J*Anonymous, 1975, B*Bourne & Fox, 1973, J*Eddy, 1977, J*Ewing, 1972 & 1978, J*Glass, 1977, J*Gordis & Sereny, 1980, B*Knott, Beard & Fischer, 1972, J*Krupka & Blume, 1980, J*Kwentus & Major, 1979, J*Lewis, 1980, J*Mathew, Clagnorn & Lergen, 1979, B*Mullin, 1978, J*Neuberger et al., 1980, J*Smith, 1984, J*Tennant, 1980, J*Wastermeyer, 1980, to spark discussions about the role and responsibility of the family physician.

In the clinic, rehabilitation and aftercare treatment planning should be done under the watchful eye of the clinical preceptor. The collaborative relationship with specialized treatment facilities also should be clinically supervised. Simulated clinical cases could be used in a seminar setting to teach treatment planning skills. Preferably this should be done with someone present from the local substance abuse referral system who can comment on the appropriateness of options. During block rotations to clinical rehabilitation programs, the student should participate in treatment planning for both the inpatient or outpatient program and for the aftercare component.

EVALUATION Treatment Planning & Referral

While in a block rotation in a rehabilitation facility, clinicians could evaluate the student's capacity to plan rational rehabilitation for patients. Or family medicine faculty could examine the capacity of the students to plan treatment for simulated cases or for real cases in the clinic.

SELF-HELP RECOVERY GROUPS

LEARNING OBJECTIVES: Self-Help Recovery Groups

The physician should be able to educate the patient and family about the benefits of attending self-help groups in the community as a part of recovery. The doctor should be able to make an effective referral to the self-help community and know how to assess and correct difficulties in utilizing these groups.

COMMENTS: Self-Help Recovery Groups

ALCOHOLICS ANONYMOUS

Alcoholics Anonymous [R*AA] is undoubtedly responsible for the recovery of more alcoholics than any other single organization. Founded in Ohio in 1935 by a surgeon and a stockbroker, two drinking "buddies" who helped each other to recover, AA is a lay organization developed by alcoholics for alcoholics. It describes itself as a program of attraction rather than of promotion. It utilizes the disease concept, and considers abstinence as a pre-

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requisite of recovery (J*Goodwin et al., 1979, J*Tournier, 1979). As stated in the AA Preamble, it is "a fellowship of men and women who share experience--with each other--to solve their common problem and help others recover from alcoholism" (Alcoholics Anonymous, 1976). Many groups have generalized the definition of alcoholism to include all chemical dependencies. Most AA chapters meet once a week for a meeting in a fixed location. But they serve their membership informally 24 hours a day, 7 days a week through informal contact between members. Some chapters do outreach to new members or old members who have relapsed (twelfth step calls), and "go on commitments" to other chapters or treatment or educational facilities. There are several types of AA meetings.

1. **Open Speaker.** Meetings which anyone may attend are designed to provide an opportunity for the public to learn about alcoholism (i.e., what is it, do its victims recover, how bad does it have to be, can AA help in recovery, etc.). In this type of meeting, one to three speakers will lecture to the group, usually with quite a bit of humor, often very entertaining, usually touching upon very personal, affectively loaded material from their lives. Often family attend open meetings with or without the alcoholic. These meetings serve to reaffirm the value of sobriety for sober members, as speakers recount their troubles from drinking ("drunkalog") and the benefits that entered their lives since quitting drinking. The humor, laughing at oneself, helps lighten the load of those in the audience who are currently struggling to overcome some tough problems. Often such meetings also provide an opportunity to celebrate anniversaries of sobriety among chapter members, reinforcing the importance of sober life style. Many open speaker groups will invite good speakers from other chapters ("going on a commitment"), providing an opportunity for cross-fertilization of ideas and philosophies about recovery, and preventing a chapter from getting stale and boring as the same speakers recur on the schedule.
2. **Closed Discussion Meetings** are open only to those who want to work on their own recovery. Usually smaller, or if large, subdivided into "tables" of 5-10 persons, these meetings offer an opportunity to discuss personal issues with a small group of others who "have been there". Typical topics raised might include, dealing with resentments, having the "blues", fighting the urge to drink, dealing with ignorance in others, confusion about how to change oneself during recovery, etc. Usually attended by persons who are actively working through the twelve steps of AA, and best when at least a couple of seasoned members are present, this type of meeting resembles group therapy except for the more variable attendance from meeting to meeting and the absence of a group therapist. Often the group leader at each table is selected by mutual consent at the start

of the meeting. One variety of closed discussion meeting is the Step Meeting. This is an aid to those who are working on the twelve steps. Usually there will be a table for each step, or lacking sufficient numbers, the steps will rotate on a weekly basis. In particular, the first step (surrender) and the tenth step (taking a fearless and searching personal, moral inventory) usually require some assistance from others and are usually found at a Step Meeting. The format is usually for a monologue to begin with one person, followed by each neighbor in turn until everyone has had an opportunity to speak, followed by general discussion until the close of the meeting.

2. Sometimes found in central urban areas, is a chapter which resembles a crisis drop-in center. It often features meetings around the clock, seven days of the week, with coffee and moral support for those who are having a rough time at the moment. It also provides a place for the idle alcoholic to find conviviality when hostility may be found elsewhere in his or her life. An opportunity to help others who are struggling by sharing one's own experiences (twelfth step) is readily available in such a setting. Though twelfth step calls are a regular part of the activities of many chapters.
4. There are numerous social and educational functions sponsored by members of the AA and Al-Anon community which are not officially part of meetings but which may be tied to them by location, time and attendance. For example, conferences featuring popular speakers on the "lecture circuit" may be accompanied by AA and Al-Anon meetings before, during or after the conference. AA/Al-Anon dances, parties and picnics often bring the families together for sober socialization, preventing gravitation towards chemical promoting celebrations in the mainstream community. Some communities have Alano Clubs with a clubhouse-meeting place where the drop-in function may be achieved, and where parties and AA/Al-Anon/Alateen meetings are held.

The AA sponsor is an important element of the program. After attending a number of meetings, the newcomer to AA is encouraged to select a member with considerable duration of sobriety to serve as a mentor to the recovery process. This person may be used as an advisor to working through the various steps of AA and as a support during the rough times. This person will often share personal experiences (self-disclosure) and cite elegans appropriate for the moment in a form of "folk psychotherapy" which comprises this function as sponsor. D'Alibrandi, 1978 & 1982; M*Zimberg, 1982.

AA may be used in a variety of ways by different people, because need and resistance varies among individuals. Some will become deeply involved in the AA community, while others will flit in and out, and still others will barely participate at all.

Some will avoid the intimate discussion meetings in preference for the speaker meetings, where one can passively attend sitting quietly without involvement. In such a case, someone might hide a psychosis, an anxiety disorder (fear of speaking in groups), dishonesty, continued drinking or drug intake, or lack of acceptance of the diagnosis. If a person does not have or use the AA sponsor by the sixth month of abstinence (after the initial "honeymoon" period has passed), the risk of relapse may begin to rise considerably. When a patient is in active treatment, it is sometimes possible for the therapist to meet with the sponsor in the presence of the patient to discuss progress and resistance. Questions about the attendance patterns, use of the sponsor, attitudes towards AA and the twelve steps may serve as a sensitive relapse predictor for the clinician who is working with the alcoholic/drug abuser. As the patient drifts away from the AA support system, he or she drifts towards the mainstream community in which sobriety was so difficult to maintain in the past. While some patients do alright without the involvement in AA, there are many who do not. And many of the people who initially have difficulties in using the AA program to assist them in recovery can be helped to constructively alter the way in which they use or relate to AA.

Some therapists and physicians are threatened by the AA involvement of their patients. The advice of the "folk psychotherapists" may not jive with the philosophy of the clinician. For example, patients may be advised to refuse medications from the doctor, especially abusable psychoactive drugs. While AA does not advocate that members practice medicine on one another, members may share "horror stories" in which physicians played a destructive enabling role, and this might frighten the patient (or the sponsor) who feels his or her situation may be analogous. Likewise, AA members may be sent or taken to detox facilities, rehabilitation programs, or other doctors or therapists in the community known for their expertise in the addictive disorders without consulting the primary care physician. Until the primary care physician establishes a reputation in the recovering community of being helpful to the recovery of the substance abuser, he or she will probably be treated with suspicion by AA and Al-Anon members. Aside from the benefit to the physician's reputation of providing excellent recovery-facilitative care to chemically dependent families, the physician can also enhance reputation by offering to speak on related topics at AA/Al-Anon functions, by supporting local treatment programs (e.g., the detox facility, half-way house, rehab program, etc.) as a consultant, board member, fundraiser, helper in setting up a new facility, or by offering to give "second opinions" to alcoholic patients going to other doctors in the community.

At AA's meetings, held in 92 countries, a cross-section of all classes of society is found. Called sobriety clubs in some countries, the details of the program vary reflecting the local culture, class structure, racial mix, etc. There are also homogeneous special AA groups for people with special needs such as physicians, lawyers, pilots, gay persons, women, young people

and non-smokers. Out of the AA self-help movement have developed a number of other groups (e.g., Al-Anon, Narcotics Anonymous, Gamblers Anonymous, Pill Heads Anonymous, Women for Sobriety, Overeaters Anonymous, Families Anonymous, Parents Anonymous, etc.). These programs are available at no cost to the person seeking help.

AL-ANON

Al-Anon is a self-help program intended for those who care about an alcoholic (S*Abion, 1982; J*Abion, 1974; R*Al-Anon, J*Bailey, 1963 & 1965; H*Estes, 1977). Similar in format to the AA program, Al-Anon encourages the member to take responsibility for personal actions (especially enabling behavior) and to cease taking responsibility for the behavior of others (especially the alcoholic). Called "disengagement", the Al-Anon member is urged to begin practicing "tough love" by refusing to run interference or mop up messes for the alcoholic who is in trouble with the surrounding environment. Many Al-Anon groups now have expanded their membership to include those who care about victims of any substance abuse. There are special meetings for young people concerned about parents with substance abuse problems (Alateen) and for adults who were children of alcoholics (A.C.A.). In general, the format and philosophy of these groups parallels that of AA. One major difference between Alateen and AA or Al-Anon is the requirement that adults from AA and Al-Anon are present as "sponsors" at all Alateen meetings. It is common for young people in Alateen to have their own substance abuse problems to deal with in addition to their issues of enabling. Experiments with younger age groups (i.e., preteens and toddlers) have spawned Alatots, but these have not yet spread beyond rehabilitation centers.

FAMILIES ANONYMOUS

When more than one member of the family has a substance abuse problem, it is advised that each attend both AA and Al-Anon or Alateen meetings to deal with both the recovery from personal addictive behavior and the recovery from enabling behavior. Another option that is helpful is Families Anonymous, in some locations known as Ala-Fam, which has sprung up due to the recognition of a major gap in recovery programs when AA and Al-Anon are used without family treatment. The individual focus of the self-help recovery programs interferes with focusing on interactional problems which arise during the illness and recovery. Families Anonymous usually meets in conjunction with AA and Al-Anon, supplementing the individual focus with opportunities to meet as multiple family groups and address some of the interactional issues.

REFERRAL

Some patients will be initially resistant to self-help, and present a variety of excuses why it will not work for them, or

why they do not like it. Common complaints include fear of public embarrassment, difficulty speaking in groups, aversion to a "religious" orientation, aversion to tobacco smoke, failure to identify with participants, desire to forget about substance abuse rather than be reminded of it, fear of recriminations for attending delivered by family members, lack of time or transportation, etc. Frequently, these are only excuses for continuing to stay sick. But these complaints also arise in patients who are otherwise working actively on recovery. Some patients do not overtly complain about AA but they procrastinate attending meetings or do not attend regularly or attend in a hostile or intoxicated state. Many people who eventually join the self-help recovery groups had to overcome this initial resistance. Therefore, the clinician should be patient and persistent, and not be discouraged by a negative response. The clinician should him- or herself attend several meetings to knowledgeably discuss the resistance and motivate the patient to attend AA. Referral can be facilitated by giving the patient the local AA telephone number and giving the patient AA literature. Even more effective would be to call the AA number or an AA member known to the clinician, and hand the patient the phone in order to set up a meeting date.

One useful strategy for helping patients and family members to better utilize the self-help recovery groups is to introduce them to INTAKE SPONSORS. These individuals reach out to new potential members, spending time with them to explain the program, making introductions to others with whom they might share values and identity, and providing transportation, suggestions and moral support as they shop around looking for the most comfortable chapters.

The family physician can and should monitor utilization of self-help. The doctor can monitor attendance by having the patient (or sponsor) fill out attendance sheets. AA recommends that newcomers attend "90 meetings in 90 days." Most patients will not do so, and an acceptable compromise might be four meetings a week. If group therapy is attended regularly in addition to attending AA, perhaps two or three AA meetings might suffice. The duration of attendance should be FOR AT LEAST TWO YEARS. Patients should be encouraged to continue attending meetings regularly beyond the initial two years.

Beyond urging continuing involvement, patients may need to be encouraged to try a wide variety of meetings to find groups that will be most useful for them at any particular stage in the recovery. And patients may benefit from questioning about their progress in the self-help program. If spiritual growth is not readily apparent, chances are that the patient is not using the program as intended. If the program is being used inappropriately, or even harmfully, the physician may need to actively encourage or counsel alterations. For instance, the person who attends 20 AA meetings per week but does not work, spend time with the family, etc. and is neither working on the steps nor obtaining any counseling support, may be abusing AA to avoid change. A person who becomes a sponsor and accepts twelfth step calls and speaking commitments early in recovery but either at-

tends no closed meetings or focuses only on others at closed meetings may be playing therapist to boost self-esteem and to avoid dealing with his or her own problems.

EFFICACY

Although few studies are available, AA appears to help from a minimum of 33% to a maximum of 90% of the people who have attended its meetings, depending of course on how often and how long these people attend the meetings (B*Bean, Whittfield & Williams, in press). For those who reach one year of abstinence with the help of at least 10 AA meetings, the probability of relapse falls to 47%, after two years it falls to 25%.

One barrier to scientifically assessing efficacy of self-help recovery groups is the difficulty in keeping track of members for a research study. Further problems arise in relying on self-report. To make matters worse, those who are lost to follow-up who first attended an AA meeting may have relapsed, gone to a different AA chapter, or have recovered using another modality. There are large numbers of such infrequent visitors to AA meetings. Nevertheless, those who continue to attend AA meetings regularly and for a long time seem to do reasonably well. But is this because they were self-selected for their preference for AA or willingness to abstain (J*Edwards et al., 1977)? Thorough scientific study of the self-help recovery programs still needs to be done (J*Glaser & Ogborne, 1982).

TEACHING HINTS: Self-Help Recovery Groups

Ask the students to list as many self-help groups for chemically dependent patients and their families as they can find in the community. Suggest that they use the telephone directory, advice from staff at local treatment facilities and crisis hot-lines, and information from their own friends and families. Compile a directory of self-help recovery resources with information about how to access each resource. Have the students attend as many meetings of different sorts as they can, contrasting their experiences as a group in a seminar setting. It might be helpful to assign each student an intake sponsor from one of the self-help recovery groups (see page 8-22).

Have them watch a film (A*AA and the alcoholic) and discuss their reactions with a panel of volunteers from local AA and other self-help recovery program chapters.

Have the students collect and familiarize themselves with literature distributed at the meetings and then present to one another the major thrust of each (B*Alcoholics Anonymous, 1976).

EVALUATION: Self-Help Recovery Groups

Ask the student to briefly describe Alcoholics Anonymous, Al-Anon, and other self-help recovery programs and the various types of meetings that are available for each. A sample response might read: AA is a fellowship of people who share their experiences, strength, and hope to recover from alcoholism or other drug problems. It is a lay organization that does not consider itself to be treatment, although it is one of the most effective aids to recovery. It uses 12 steps to recovery, and 12 "traditions" as guidelines for operation. Its meetings are free and widely available. Members are anonymous and no records are kept. Open meetings are for anyone. Closed meetings are for those with a desire to stop drinking (or taking other psychoactive abusable drugs). "Step meetings" discuss the aspects of the 12 Steps. Beginners meetings are for newcomers.

Have the student discuss the basic thrust of the 12 Steps of AA. A sample response might read: The 12 Steps were developed in the late 1930s to help provide a path to long-term recovery from alcoholism. Summarized, they include (1) admission of a drinking problem; (2) changing isolation to sharing, and recognition of a power greater than oneself; (3) self-examination, catharsis, and personality change; (4) improved relationships; and (5) helping others [B*Anonymous, 1977; B*Bean, Whitfield & Williams, in press].

Ask the student to briefly describe how to make a referral to AA or other self-help recovery program. Or better yet, observe the resident making such a referral. Have the student discuss advantages and disadvantages of AA. A sample response might read:

ADVANTAGES: AA is free, widely available, welcoming, warm and supportive, and is effective. The degree of respect offered to a person who wishes to recover is unmatched anywhere else in the world. This respect includes an understanding that the person needs to design and execute his or her own recovery program. The initial responsibility may be too much for some, but eventually when recovery has begun, the person will have gained self-respect for having achieved the progress so far.

DISADVANTAGES: Some people complain of a religious overtone to the program. However, actually AA is not religious, rather it is spiritual in orientation. References to a Higher Power is to be interpreted as the individual understands it. This may mean nature, mankind or the AA program for some, while for others it may refer to Jesus, God, Bhudda, etc. Since AA and the other self-help recovery groups do not discriminate by religious preference, members who are offended by inclusion of references to Christian theology may either do what they do when it happens in other contexts (i.e., ignore it, translate it, or ask the group to speak more generically).

Some people complain of repetition, though many people need it. Such complaints suggest patient stagnation, passivity and a wish to be spoon-fed recovery. When the patient actively participates in the self-help recovery groups, the repetition will be applied to the current situation differently each time the same message is received, because the patient always will be changing.

The lack of formal assignment of members to "therapists" and the informal structure of the organization produces an easy net to slip through when a person is not motivated to recover. Intake sponsors and outreach by concerned members counteracts this drawback to some degree, but there is no substitute for self-motivation.

RELAPSE MANAGEMENT

LEARNING OBJECTIVES: Relapse Management

The student should be able to educate the patient and the patient's family about the chronic relapsing nature of the disease. The student should be able to recognize the common precursors of relapse clinically anticipate relapses so that some may be averted before they occur or the severity can be minimized. The resident should be able to help the chemically dependent patient and family develop strategies to prevent relapse. When one occurs, the resident should be capable of helping the family discover the reasons for the failure of the recovery plan and aid them in modifying the continuing recovery program to address the unmet needs discovered around a relapse.

COMMENTS: Relapse Management

Alcoholism and other chemical dependencies should be recognized as fitting into the chronic disease model. These are bio-psycho-social illnesses that affect not only chemically dependent people, but their families as well. The tendency to relapse (or "slip") is very high, and constantly present, no matter how long a person has been chemically free. Relapses or slips are more common in the first few weeks, and tend to occur less frequently as the length of recovery increases. Independence from chemicals for two years is considered a benchmark; after this period, relapse is much less common, but always possible.

HIGH-RISK SITUATIONS

The physician needs to be aware of feelings, thoughts and behaviors that trigger relapse. A patient may have euphoric recall (i.e., may only remember the benefits of alcohol or other

drugs, but may ignore the negative effects. Memory impairment, both organic and functional, may exist. Patients in recovery often engage in self-flagellation (guilt, shame, and remorse) about previous failures to control substance ingestion. These feelings may trigger a relapse resulting in a self-fulfilling prophecy about not being able to remain sober. Depression, pain, anxiety, and insomnia may also lead to relapse if the chemically dependent person uses psychoactive drugs to treat these symptoms. Physicians may inadvertently prescribe medication for these symptoms, and the patient may ingest them, not realizing that it may enhance the risk of relapse.

After periods of sobriety, the chemically dependent person may resort to magical thinking about the ability to control drinking or other drug use, and trial usage may result in relapse. The family may encourage this.

The family of the chemically dependent patient has an entire repertory of behaviors that they learned during the period of active use. When the behaviors change during recovery, a new set of family behaviors may be appropriate. Even though the family had achieved a degree of homeostasis, this homeostasis was pathological, and change may be difficult. The resultant resistance to change may be actions or behaviors that intentionally or unintentionally encourage relapse. An example of such a situation is the spouse who had assumed responsibility for the family finances, and now would be very reluctant and resentful about losing control of that important function.

The early recovering patient may experience periods of an almost euphoric state, and may receive a great deal of positive attention. However, the family members may only focus on the past harm and violated trust, and may expect that this could happen again. As a result, family members may reel and express jealousy, rage, and skepticism. The conflicting experience of patient and family can and often does interfere with the recovery of the patient.

Another predictor of relapse is the insistence by a recovering patient on continuing relationships with other chemically dependent family and friends, or socializing in the places where chemical use was common before recovery. Fueled by the desire to maintain stability in as much of life as possible despite the pressure to change exerted by rehabilitation staff and self-help recovery group members, the patient may appear "cocky" about the belief that drink or drug refusal skills will suffice to avoid relapse in this high-risk environment. Although chemically dependent patients need always to be reminded that sobriety is their responsibility, certain outside negative influences have a potential to interfere with recovery.

Return to life in the real world may lead the alcoholic or addict to neglect sobriety and personal needs. An acronym used in AA to remind one of some of the universal high-risk situations can be a helpful.

- H HUNGER
- A ANGER
- L LONELINESS
- T TIREDNESS

Alcoholics often give the following as factors in their relapse: (1) unpleasant mood states (i.e., sadness, anxiety, anger or depression), (2) external stimuli for euphoric recall (e.g., passing a bar or familiar haunt), (3) lessened cognitive vigilance (e.g., thinking "one drink will do no harm"), and (4) social anxiety. Other specific triggers may be recognized in certain people.

RESPONSE TO RELAPSE

Relapse management may require investment of a great deal of time and energy. The physician should be aware of the resources available for recovery, and may wish to refer patients back to their specialized treatment professionals who had been working with them prior to relapse, while maintaining a positive and supportive stance towards the patient and family. Usually the patient and family will feel quite bad about the relapse, though behavior may not necessarily reflect that (i.e., the patient may act obnoxious and cocky to cover up the sadness and depression). It is important for the physician, family member, or other close person to express disappointment when relapse occurs, because indifference, lack of concern, or overt hostility will only reinforce the hopelessness the chemically dependent person feels. But it is also important to explain that one is angry at the person's disease and sad about the inadequacy of the previous recovery plan.

Understanding the recovery process and its chronic nature will enable the physician to reassure the recovering patient that what is being experienced is to be expected. Educating the family about the value of the relapse as an opportunity for growth and reexamination of the recovery plan, anticipating their resentment and lack of trust, and reinforcing their need for their own recovery is also important. Relapses or "slips" may at times be therapeutic if the physician constructively uses them as examples of weaknesses in the recovery plan rather than weaknesses in the person. Hence, a relapse might be used to reinforce the importance of abstinence to a patient or family who advocated controlled drinking. Or it might provide an opportunity to increase reliance of the patient or family on outside support (e.g., AA) whenever such magical thinking begins. Or a relapse might be the needed crisis to motivate the patient or family to reconsider previously rejected rehabilitation recommendations. Reinforcing the need for a sponsor in Alcoholics Anonymous, one who has experienced many of the same feelings and behaviors, can be extremely helpful in preventing further relapse.

TEACHING HINTS: Relapse Management

Interviews with recovering patients with the specific objective of obtaining information about the triggers of their own relapses is an excellent way of building a knowledge base of how the addicted person gets snared in relapse. In addition, attention should be paid to the alterations that were suggested in the recovery plan following the relapse. Attendance at AA and Alanon meetings will also impart some knowledge of this and is likely to also teach the student to respect the relapsing family more fully.

In a classroom setting, films like *Talking Back*, *The dry drunk syndrome*, *A*If you loved me, A*I'll quit tomorrow*, *A*Our brother's keeper*, *A*The enabler* can be used to portray the complex nature of the relapse process from a family perspective. Have the students watch for triggers and analyze the constructive and destructive responses of all involved.

In clinic when a relapsing patient is encountered, use the metaphor of chronic diabetes to teach about the most appropriate physician attitude towards the relapsing family. Watch for expression of frustration or fear by the clinician, because it may result in overt hostility or neglectful clinical decisions which result in harm of the patient or family. Have the resident write an analysis of the reason for the relapse and a modified recovery plan to prevent the same trigger from activating relapse again.

EVALUATION: Relapse Management

Critique modifications of recovery plans of relapsed patients using the criteria that it should be realistic and should address the triggers of the current relapse and all previous ones.

CHAPTER 7: PREVENTION OF SUBSTANCE ABUSE PROBLEMS

By Michael R. Liepman & Ted D. Nirenberg

OVERALL OBJECTIVES

The family medicine instructor should be able to teach the residents and students about the models and strategies of primary, secondary and tertiary prevention, their effectiveness in reducing the prevalence and severity of substance abuse and its consequences, and the pros and cons of each. Furthermore, the instructor should be able to assist the residents and students in discovering appropriate roles for themselves as physicians who participate in substance abuse problem prevention efforts. Through the precepting process, the instructor should be able to help the residents develop skills in screening, high-risk counseling, and early intervention to improve the overall health of their patients.

COMMENTS: Prevention

Primary prevention is designed to lower the risk of onset of an illness in a population. Secondary prevention selectively finds risk factors and cases of the illness in individuals early in its course and intervenes to prevent progression and thereby reduces total cumulative harm. Tertiary prevention selectively attempts to reduce the severity of harm in later stages of the illness through restorative and rehabilitative care to make the best of what resources remain. Health promotion attempts to assure that individuals and the population as a whole have maximal opportunity to achieve potential, in physical, psychological, social and spiritual arenas [B*Hess, 1983].

Ethics of Prevention

Prevention is a public-health strategy used to reduce a population's risk of suffering damaging consequences. When applying preventive measures to a behavioral disorder, to reduce risks is in the best interest of the population, hence prevention has a benevolent purpose for the society as a whole, but the payoff may not necessarily benefit each individual in a direct way. Prevention may produce some small social benefit, but the strategies may rather significantly abridge personal freedom. The physician who intervenes to prevent harm in a patient or family may be seen as taking a paternalistic approach to health which denies people the right to put their own health at risk. Yet there are components of Western society which support personal security (through social welfare, health benefits, charitable donations) to people who have taken these risks and lost their resources. The degree to which individual freedom is to be tolerated at the cost to the rest of taxpayers who will foot the bill is currently

a hot topic of debate. Witness the progressively more negative attitude towards drunk drivers and the community movements to toughen laws and enforcement. Hence, the balance of costs versus benefits when selecting prevention strategies is crucial.

LEARNING OBJECTIVE: Primary Prevention

The resident should be aware of the current information about how alcohol or drug abuse or dependence can be prevented [B*Collins, 1978]. Because of etiologic uncertainties about the addictive disorders, the resident should be familiar with the differing philosophies behind current efforts and should understand the positive and negative implications of the various approaches.

COMMENTS: Primary Prevention

Primary prevention consists of strategies used to avoid the onset of an illness in a population. For each of the addictive disorders the exact etiology (or etiological factors) is (are) unclear. While much is known, there are more questions than answers. There are five major approaches towards primary prevention [B*Messolonghites, 1977; B*Miller & Nirenberg, 1984; B*Robinson, 1982; B*Smart, 1983]:

1. consumption control in the entire population,
2. alteration of cultural norms and attitudes towards patterns of use,
3. alteration of parent-child and peer-peer interactions concerning abuse,
4. offering alternatives to chemical use and abuse, and
5. counseling caution in selected high-risk cases.

PREVENTION VIA CONSUMPTION CONTROL

LEARNING OBJECTIVES: Consumption Control

The resident will understand the consumption control hypothesis of alcoholism prevalence and the implications this has for public health policy. Furthermore, the resident will understand the limitations of public policy initiatives in controlling human behavior.

COMMENTS: Consumption Control

The consumption-control model attempts to prevent the increasing incidence of chemical dependency by limiting consumption of the entire population. Ledermann [B*1956; J*Popham & Schmidt, 1978; B*Rankin et al., 1975] challenged conventional wisdom when he proposed that a unimodal rather than bimodal curve could be

drawn to describe the alcohol consumption of any population (see Figure 7-1). He claimed that once a population's average consumption point was measured, the standard curve would describe accurately the total distribution of consumption. This shattered prior assumptions that alcoholic individuals represented a qualitatively distinct population whose drinking patterns bore no resemblance to that of the normative society [B*Pattison, 1984] (15% of adults consume 74% of the alcohol [B*Mandell, 1982]). Furthermore, it has been suggested that lowering or raising the average consumption point in a population will alter the incidence of alcoholism and its consequences [B*Druun et al., 1975; J*de Lint & Schmidt, 1971; J*Duffy & Cohen, 1978; B*Popham, Schmidt & de Lint, 1976; B*Schmidt, 1977; B*Schmidt & Popham, 1978; J*Skog, 1980]. The basis of this model is the correlation of cirrhosis mortality rates at various times and locations with the consumption rates of alcohol (see Figure 7-2). Further evidence of support utilized hospitalizations in Finland against longitudinal data over a six-year interval during which per capita drinking doubled [J*Poikolainen, 1983].

Critics of the consumption-control model cite the need to correlate alcohol-induced consequences other than cirrhosis mortality with the consumption rates before concluding that alcoholism rates are determined in this way [J*Parker & Harman, 1978; J*Schmidt & Popham, 1978]. Poikolainen [J*1983] has begun to do this. A similar complaint can be made against this model when it

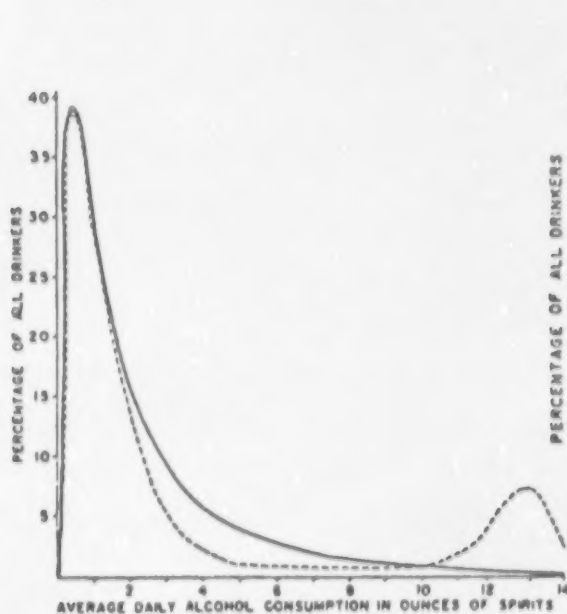


Figure 7-1: Bimodal versus unimodal consumption models^a

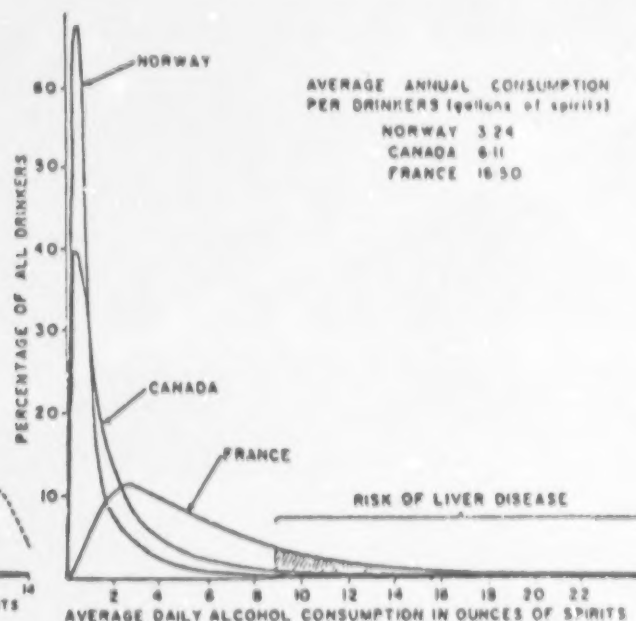


Figure 7-2: Consumption distribution curves of three nations^a

^a Reprinted with permission from [B*Kalant & Kalant, 1971]

is used to predict consumption distribution of other drugs. Furthermore, data must be gathered looking simultaneously at all psychoactive substances because of a continuing trend towards shifts in utilization from one drug to another as the supply, price, and legal risks are altered. Aside from scientific considerations, public officials are reluctant to take the unpopular stand of trying to reduce overall consumption, by the entire population, of popular drugs (e.g., alcohol, tobacco, caffeine, marijuana) lest they offend the producers and the users among the voters, campaign contributors and lobbyists [J*Cavanagh & Clairmonte, 1983; H*Jacobson, Hacker & Atkins, 1983; J*Selvaggio, 1983]. They would prefer to see addictive disorders as a special problem limited to a minority among the population whose misery could be dealt with restoratively.

Schmidt and Popham [B*1978] note that the relative price of beverage alcohol to other consumer items and to average disposable income has been falling in direct correlation with increases in consumption in Ontario and in other nations. In particular, distilled spirits (the most concentrated alcoholic beverage) have dropped in price the most. They suggest a tripartite public policy strategy that would: (1) adjust, through liquor taxation, the prices of alcoholic beverages to keep them constant over time relative to the average disposable income of consumers, (2) require that all new proposals to loosen governmental controls over alcoholic beverages be scrutinized in terms of their public health impact, and (3) provide government sponsored public education programming to alert the population to the personal and community health and socioeconomic consequences of heavy alcohol consumption. Some experiments with alteration of availability via legal restrictions and/or taxation have demonstrated effectiveness in reducing consumption [B*Brunn et al., 1975; B*Gotestam & Rostum, 1984; B*Pattison, 1982; B*Sytnsky, 1984], but the cost to personal freedom and the difficulties in enforcement must be considered (c.f. prohibition in the U.S.). The impact of alteration of the average rate of alcohol consumption on alcoholism incidence rates is still unclear [B*Wilkinson, 1970].

For illicit drugs, tough law enforcement campaigns have been tried in an effort to reduce supply and increase the risk and cost of distribution of drugs [B*Durrin, 1978]. The result has been largely disappointing. While an impact has been observed, it has often been paradoxical. For instance, Governor Rockefeller's 1973 crackdown in New York state led to the following: "prices increased, more dealers and users carried guns, and the quality of heroin decreased" [B*Ray, 1978, p. 46]. Some effective interventions such as the ten-fold reduction in legal production of amphetamines resulted in an almost total disappearance of amphetamines from the street supplies. However, user demand led to dealer substitution with caffeine, phenylpropanolamine, cocaine and other similar drugs which were sold under the guise of amphetamines [B*Morgan & Kagan, 1975]. However, when a drug can be simply manufactured illicitly, the result of reduction of legal sources is a shift to illicit production with a concomitant reduction of quality control and usually an increase in price. As

the prices increase, the difficulty of enforcement increases because of bribery, bail-jumping, and legal games in the courts. As drug utilization or drug arrests increase, the prevalence of successfully adjudicated cases decreases as the courts and prisons pile up backlogs. Ray [B*1978, p 46] summarizes the issue: "tough drug laws will not work and are not enforced because they are not supported psychologically or fiscally by the public or by many members of the law enforcement or criminal justice system." We have learned this lesson from cannabis [B*Erickson, 1980].

Applying the consumption control model to cigarette smoking has led to the popularity of low tar and nicotine yield cigarettes. These cigarettes are essentially the same in content as regular ones except that they have various filters and air-pores in the paper which lead to dilution of the mainstream smoke and partial condensation of the tars in the filter. Doubts as to the efficacy of the low tar and nicotine cigarettes have been raised by measurement of toxins in the bodies of smokers using them; carbon monoxide and nicotine levels may not be significantly lower due to smoker compensation by [J*Korolowski et al., 1980 & 1982]:

1. inhaling sidestream smoke,
2. by increasing puff depth and frequency,
3. by smoking more cigarettes per day,
4. and by placing fingers or lips over the air-pores.

A number of examples of partial prohibitions have been tried when total prohibition seemed either impossible or unreasonable. Age restrictions are imposed on alcohol and tobacco consumption in the United States, though poorly enforced. Cigarettes are sold from vending machines which cannot attest to the buyer's age, and alcohol is sold only by people, but it is easy for young people to find someone old enough to purchase it for them. In the past, cultural values prohibited most women from drinking or smoking. This has nearly disappeared in recent years due to political equalization of the sexes and vigorous advertising campaigns by producers. "Blue laws" have been used to restrict the hours of alcohol consumption by closing bars and liquor stores at certain times of the week. Similarly, there have been "dry" counties throughout the nation that prohibit the use or sale of alcohol.

One type of partial prohibition is restricting location of use. People are restricted from using alcohol and smoking in certain buildings such as schools and hospitals, or in certain locations within buildings or vehicles, such as offices, airplanes, sections of theaters, restaurants, etc. Once again, enforcement problems become an issue. One benefit of these restrictions are to the innocent bystanders. It is important to realize that the chronic carbon monoxide levels of a smoker often surpass those set by OSHA as maximum safe levels. It has become well established that sidestream smoke from cigarettes affects the health of those breathing from the same source of air [J*Aronow, 1978; J*Hirayama, 1981; J*White & Froeb, 1980]. Furthermore, the sidestream smoke from the newer "healthy" low tar and nicotine cigarettes appears to be more dangerous to bystanders than the

higher yield cigarettes, probably because the smoker gets a diluted version of the smoke through the filter mechanism while the bystander gets the smoke straight. Some smokers compensate for their low yield by increasing the frequency of cigarette consumption, to the dismay of bystanders who do not wish to smoke. Recent tightening of regulations about occupational health through litigation has improved the situation by forcing employers either to restrict smoking or provide improved ventilation.

Taxation of popular psychoactive substances has been going on for centuries. While taxation may reduce consumption by increasing the cost of these substances, its primary purpose usually has been generation of income for the government. The result of such tax policies has typically been civil disobedience, smuggling or switching to another substance [B*Ray, 1978].

Another method of controlling substance ingestion is to limit access to it through a "gatekeeper" [B*Nightingale, 1978]. Physicians are asked to play this role with prescription drugs. It is hoped that their ethical and medical training will lead physicians to make wise decisions with regard to distribution of these drugs to the population. However, physicians have become the targets of pharmaceutical houses wishing to market their products. Impaired or unethical physicians often become identified as easy targets for drug-seeking patients. And many addicted physicians believe that many of their patients need psychoactive drugs in order to live comfortably and function effectively.

Ironically, while research findings from studies supported by the United States Government clearly demonstrate the health hazards of tobacco smoking, the government continues to subsidize the tobacco industry in numerous financial and regulatory ways. These include: (1) federal financial support for tobacco growers; (2) purchase of tobacco products to be sent abroad as foreign aid; (3) subsidy of military tobacco sales; and (4) protection of tobacco products from health and safety regulation that are applied to other drugs. For example, efforts to increase hazard labeling on cigarette packages and advertisements have been resisted by governmental agencies influenced by lobbying efforts of the tobacco industry. Likewise, efforts to introduce legislation requiring all cigarettes sold to be automatically self-extinguishing if not puffed on regularly have met great resistance. Political pressure exerted by the tobacco industry exemplifies the power of financial interests in the federal bureaucracy to overturn logic and to overshadow ideals.

Family physicians may be considered by local politicians to be wise and knowledgeable about matters concerning substance abuse problems. They may seek advice when considering legislation to alter controls on psychoactive drugs. As one who sees the clinical side of these problems, one might also wish to join in the political process. For instance, in many communities there is burgeoning public and political interest in the tragedies caused

by drunk drivers. The local physician may become involved in community interventions to reduce the risk of drunk driving. While the family physician is undoubtedly quite busy, involvement in community affairs of this sort enhances the physician's reputation in the community, improves the community in which the physician lives and works, and provides for the community a perspective on the complex issues involved in solving some of these problems which may not be available from any other member of the community.

TEACHING HINTS: Consumption Control

There have been numerous governmental attempts at the prohibition of the use of the following drugs in the United States and elsewhere: (1) alcohol; (2) marijuana; (3) heroin; (4) cocaine; (5) LSD and other hallucinogens; and (6) coffee. Select a substance on which to focus during a group exercise, in which there are three subgroups: (1) those who strongly favor the prohibition of the use of a particular substance; (2) those who strongly advocate the use of the substance; and (3) those who advocate moderation of use. Have each subgroup meet for 10-15 minutes to plan a strategy for accomplishing the discussion goal. Then reconvene the larger group to discuss the ways they would interact over enforcement. If there is time, begin the exercise by viewing [A*It all adds up, 1979; A*Keep out of the reach of adults, 1974].

Have the students read about the Boston Tea Party, and the prior history of the British prohibition of coffee consumption [B*Ray, 1978], how the switch to illicit coffee provided rebellious colonists a means of avoiding payment of the British tea tax and registering a protest against taxation without representation. Have them compare the rebellious spirit in pre-revolutionary America to the "drug culture" of the sixties as it relates to the use and availability of marijuana today. Or have the students read about the history of regulation of opiates and contrast it to the history of controls over caffeinated drinks [B*Brecher, 1972; B*Ray, 1978]. Set up a debate on whether the drinking age limit for alcoholic or caffeinated beverages in your state should be changed to the average age of trainees in the class.

Have the students read about the three periods of multi-state prohibitions and the U.S. Prohibition of alcoholic beverages [B*Ray, 1978]. Then discuss the role that governmental social policy had on human behavior, both intended and unintended, and the ultimate factors which led to its eventual repeal.

Have a group of male and female students design a cultural prohibition strategy to prevent the fetal alcohol syndrome [J*Kaminski, Rumeau & Schwartz, 1978; J*Little, Streissguth, & Guzinski, 1980; J*Rosett, et al., 1978 & 1980; J*Streissguth et al., 1980]. Take into account that many of the teratogenic effects of alcohol on the fetus occur prior to a woman's awareness that she is pregnant. Have them consider the political rami-

fications of their ideas from a feminist perspective, a civil liberties perspective, a socioeconomic perspective representing the costs to the taxpayers of handicapped FAS victims, a public health perspective concerning the rights of the unborn child. Prior to the exercise, assign readings about the British gin epidemic [B*Ray, 1978] and view the slides on FAS [A*Rosett, 1981].

Offer the scenario that marijuana has just been changed to a prescription drug. Divide the group into subgroups representing (1) the manufacturers and marketers of "legal" marijuana; (2) the medical community; (3) the consumers; and (4) the law enforcement community (i.e., DEA, FDA). Have each subgroup meet for 10-15 minutes to discuss how they will accomplish their goals. Then reconvene the large group to discuss their interactions.

Discuss governmental strategies to alter consumption of tobacco products through changes in local and/or federal taxation policies. Divide the group into several subgroups in order to discuss the various sides of this issue. Groups could represent (1) the tobacco producers' lobby; (2) the consumers' lobby; (3) a lobby representing the victims of smoking diseases and their families; (4) the health profession's lobby; (5) the legislators; (6) the retailers; and (7) the law enforcement community.

EVALUATION: Consumption Control

Does the student understand the concepts of the consumption control model? Does the student understand that this is only a model which has not been proven to be true, beyond doubt? Does the student understand that public policy cannot always control human behavior?

PREVENTION VIA CULTURAL CHANGE

LEARNING OBJECTIVES: Cultural Change

The resident will understand the importance of cultural change in alteration of substance use patterns at the population level. Furthermore, the resident will be aware of the influence of mass media advertising upon the culture and human behavior.

COMMENTS: Cultural Change

Culture dictates attitudes and ritualistic behavior towards the world around us [B*Heath, 1982; B*van der Hart, 1978]. There are culturally determined rules for human relationships with psychoactive substances which dictate who may use them, when they may be used, in what manner they are to be used, and what is done with those who deviate from the norm [B*Gordon, 1978; B*Heath & Cooper, 1981; J*Heath, Waddell & Topper, 1981; B*Pittman & Snyder, 1962; B*Tseng & McDermott, 1981; B*Weil, 1972]. These cultural influences have produced marked differences in drinking

patterns, the meaning of these patterns, and the prevalence of drinking problems between different ethnic groups in the United States [B*Cahalan, & Cisin, 1976; B*Cahalan, Cisin & Crossley, 1969] (see Chapter 3 for details). For example, most people of Jewish heritage are trained to use wine in small quantities as part of religious ceremonies and that drunkenness is inappropriate behavior [B*Heath, 1982; B*Snyder, 1958]. Most Jewish people drink but few suffer from alcoholism. When someone does, he or she is the subject of much stigma. Moslems, Mormons, Jehovahs Witnesses and Southern Baptists are taught that alcohol is not to be used at all [E*Davidson, 1978; E*Pittman & Snyder, 1962]. When they do drink, they may find themselves socially alienated from their religious community. People of Irish background are taught that men may drink heavily, they may use drink to help them to express emotions which they would otherwise be reluctant to express, but violence which accompanies drunkenness is not to be directed towards one's wife or children [J*Ablon & Cunningham, 1981; J*Stivers, 1978]. More Irish men than women drink and drink heavily [B*Cahalan, Cisin & Crossley, 1969]. People raised with Italian, Portuguese and French heritage are taught that wine is a part of every day life, as a beverage with meals. Italians and Portuguese abhor drunken behavior and hide it from those outside the family [B*Lothi et al., 1958]. Striking out at members of the family while intoxicated is tolerated as behavior necessary to discipline those who deviate from family norms [J*Ablon & Cunningham, 1981]. Frenchmen often go through life without ever becoming intoxicated, but they suffer a very high prevalence of cirrhosis. Drunken behavior is acceptable in certain French settings and men are encouraged to drink to demonstrate virility [B*Wilkinson, 1970]. When Finns drink, they tend to be quite violent. Native Americans differ by tribe in drinking patterns too. The Hopi tribe members drink in a continuous pattern while the Navajo are binge drinkers. Hopi tribesmen suffer very high rates of cirrhosis; in contrast, Navajos have extremely high accidental mortality [B*Heath, 1982].

America, the great melting pot of cultures, turns into a veritable cultural can of worms when it comes to alcohol. It becomes even more complicated when other drugs are considered. Cultural rules develop over several generations after a drug is introduced into a culture. In the initial phase of experimentation, the social group gathers information about the effects of the drug, its benefits and dangers. Then consensus develops over the safest and best ways to use the drug [B*Weil, 1972]. In the U.S. there is a constant infusion of conflicting ideas about how to use a drug through cross-ethnic intermarriage, geographical mobility and electronic mass communication. In addition, we are plagued by the addition of new drugs to the marketplace every year, and their production and distribution becomes widespread before sufficient time has passed for cultural norms to be established. Family rituals about how to use drugs are passed down from ancestors, and may generate conflicts between in-laws if the rituals differ significantly. This may lead to ambivalence within the family towards drinking alcohol. Family rituals which permit uncontrolled and disruptive drinking tend to beget more cases of alcoholism in subsequent generations than rituals which exclude

drunken behavior [Wolin et al., 1979 & 1980]. And families in which there is ambivalence may be unable to develop a consensus about which rituals to permit and which to exclude [E*Liepman, Wolper & Varquez, 1982].

Efforts to influence the culture of an entire population in order to alter patterns of drug use, and therefore affect patterns of abuse, have been successfully achieved with opium, through repression of personal freedom, in the People's Republic of China through the so-called Cultural Revolution.¹ In the United States, such a dramatic change in drug use is unlikely because of the cultural heterogeneity of the population and the high degree of personal freedom which is valued. However, there have been recent changes in patterns of substance use among certain U.S. groups (e.g., women smoking and drinking more heavily, use of marijuana and other hallucinogens by the middle class, use of stimulants by athletes [B*Mandell, 1976]). Cultural change is also apparent in the shift in public opinion from denial of the health risks of smoking to acceptance of them and in the fitness subculture which has supplanted the drug subculture of the sixties.

Probably the best examples of cultural change in relation to substance use patterns are the successes of substance marketing. For instance, the alcohol and tobacco advertizers joined the women's liberation movement to increase sales by liberalizing drinking and smoking mores for women (e.g. the Virginia Slims "You've come a long way baby!" campaign). They kept the previous tough image of the "Marlboro Man" that had been so successful in marketing cigarettes to men in the first half of the twentieth century and created one palatable to female consumers by marketing new brands just for women. Now, the only portion of the consumer market which is rising is adolescent girls. Cultural shifts in tobacco usage has been notable over the last four centuries, from pipe smoking to snuff to chewing tobacco to cigar smoking to cigarette smoking. These changes were in part due to marketing strategies and in part to changes in the nature of human existence. For instance, there was no problem with spitting in the wide open West from atop a horse or while working in a field in the great plains, but spittoons in the urban offices were not well liked by gentler types. Concern over fire hazards in Boston sparked an ordinance prohibiting smoking on public streets from 1798 to 1880 [E*Ray, 1978]. Currently, the increasing restrictions on public smoking has stimulated marketing of snuff and mouth tobacco pouches.

The importance of business profits and media advertising in cultural change should not be underestimated. The annual U.S. advertising budget of the tobacco companies in 1974 was \$307 mil-

¹ The British introduced opium into China 200 years ago as a profitable import business. A prior attempt by China to rid itself of its opium problem in 1839 led to war defeat by Britain [B*Outchlerlong, 1970; B*Yen, Deely & Kaufman, 1978].

lion. In 1981, the U.S. beverage alcohol advertising costs were over \$1.1 billion [J*Cavanagh & Clairmonte, 1983; B*Ray, 1978]. Substance advertising to consumers and physicians has a major influence on the American and world culture making DuPont's motto: "Better Living through Chemistry" a truism of the western way of life. In addition, the media has supported this expanding role of chemical mind alteration by speeding the spread of new drugs. For example, the mention in a campus newspaper that smoking banana peels provided a new psychedelic "high" spread like wildfire until it was discovered to be a hoax [J*Boszetti, Goldsmith & Ungerleider, 1967]. Without extensive financial resources, it is difficult to mount a major media blitz for a long enough time and to reach a broad enough audience to have a significant impact. In 1973, one study showed that during prime time on television, public service announcements warning about dangers of alcohol were overshadowed 20-fold by alcohol commercial advertising and 40-fold by depiction of drinking during programming [B*Mandell, 1982]. There were no warnings in the print media in contrast to 34 pages of commercial advertising, but this was a time when advertisers could intimidate editors of the periodicals to suppress articles on dangers of their products. Only big businesses, large organizations and governments have large enough resources to make a significant impact [J*Cavanagh & Clairmonte, 1983; B*Jacobson, Hacker & Atkins, 1983]. For example, in the early 1970s, a highly emotional campaign of television anti-smoking counter-advertising was associated with a significant (but not substantial) reversal in market growth. Out of fear of continued damage to their market, the tobacco industry welcomed the ban on television advertising of cigarettes which they had previously fought. This also spelled the end of the counter-advertising, which had been given "equal time". The tobacco companies then spent the same amount on billboards and magazine ads, which need not provide "equal time".

Swinehart [B*1981; cited in B*Hewitt & Blane, 1984] postulates that for media messages to significantly influence people, eight sequential stages must occur: "(1) opportunity for exposure to the message, (2) actual exposure, (3) attention to the message, (4) learning the message (knowledge), (5) acceptance of the message (attitude change), (6) motivation to act, (7) recall of the message at the time of action, and (8) opportunity for action (behavior change)". In addition, the message must be palatable to the audience and social reinforcement in the community generally helps to influence this message. For example, the combined impact of public and government supported electronic media messages, billboard and bumper sticker messages, and print media articles about alcohol related motor vehicle accidents and about increased law enforcement efforts which are being undertaken, is far more likely to succeed than a single message from a single source [B*Hewitt & Blane, 1984]. Warner [J*1981] analyzed retrospectively the anti-smoking campaign over the last 30 years and found that the pronouncements by the Surgeon General of the U.S. in 1973 that cigarettes are dangerous to health and the ruling of the U.S. Supreme Court that resulted in local restrictions on public smoking may have contributed about 1% per year to the fall in per capita consumption of cigarettes.

It is important to heed the warning [J*Abion & Cunningham, 1981] that prevention efforts be mounted through or at least with the cooperation of the social institutions which play a role in perpetuation of families. It is possible that the prevention message will generate a negative or backlash reaction because it conflicts with cultural values and beliefs which are strongly held and are supported by local social institutions. For instance, if a "responsible drinking" campaign was mounted in Salt Lake City, it would probably be maligned by the Mormon religious community because it conflicts with religious doctrine and attitudes towards drinking. If prior support is sought from community institutions, it may take the form of endorsement, helping to design the message to match community needs and styles, or provision of other supportive resources.

Evaluation of an effort to change the culture is difficult because it is impossible to isolate a population from extraneous influences (e.g., legal drinking age, penalties for drunk driving), and because changes occur spontaneously over time. If it is not necessary to determine the degree to which a change can be attributed to the local effort, then it is possible merely to observe the target behavior in a well-selected sample of the population at two or more comparable times. It is not as accurate to sample self-reports as it is to sample behavior, because people may report what they think you want to hear. For example, it would be more realistic to sample breath alcohol levels on drivers stopped while driving at a barricade than to ask drivers whether they drive while intoxicated. Likewise, urine toxicology samples from players on the football team would be more accurate than asking the coach or the team members how often they use drugs.

TEACHING HINTS: Cultural Change

In a seminar setting, ask the students to devise a media campaign to encourage their patients to cease smoking or avoid driving while intoxicated (A*Ah! Relief: Responsible drug use; A*Alcohol roulette; A*Your health and alcohol). Include instructions to remain practical working on a scale that is consistent with the types of resources that a family physician might have available (e.g., being invited to talk on a popular local talk show on the local radio or television station; signs posted in the office; having health class students make posters for school hallways or for local storefronts). Discuss backlash problems that might arise from the efforts selected. If it is possible, encourage residents to actually carry out the prevention campaign in the local community. Discuss ways that the baseline of the population might be determined prior to the prevention campaign. Then discuss how a follow-up evaluation might evaluate the effectiveness of such a campaign. Be careful to pay attention to the influence of extraneous changes in the culture due to the passage of time and population targeted efforts at the state or national level. For example, how might the residents evaluate a campaign of caffeine warning posters in the clinic waiting room before and after the campaign?

EVALUATION: Cultural Change

Ask the resident to explain the role that mass media advertising plays in changing the behavior of the masses. How do the producers and marketers of psychoactive substances convince us to give them our money? How might the health professions combat the epidemic of substance abuse which is occurring through cultural change strategies?

INTERPERSONAL INNOCULATION

LEARNING OBJECTIVES: Interpersonal Innoculation

The resident will understand the interpersonal inoculation model of substance abuse prevention. The resident will be aware of the developmental implications of this strategy during adolescence. Furthermore, the resident will have clarified and tried appropriate ways to utilize this in the clinical family practice setting and in the community.

COMMENTS: Interpersonal Innoculation

Training young people to say no to drugs, alcohol and tobacco has been the focus of some prevention efforts. Values clarification, education, and assertiveness training are elements of this approach which attempts to inoculate youth who have not yet experimented with these substances so they will resist the temptation when offered by peers, parents or pushers. Adolescence is a time in life when a child begins to test his autonomy from parents by trying to master the world around him without parental support. There is naturally a withdrawal of communication, trust and dependence upon parents which is initially replaced by investment of trust and dependence on peers and extra-parental adults (e.g., coaches, teachers, employers, clergy, drug pushers, neighbors, parents of friends, relatives). These people are in a position to support the youth or lead him astray, until he reaches maturity and can decide for himself what is best. Some families fear dangers to the extent that they smother the child's attempts to be autonomous by rigid restriction, which either forces the teen to rebel or delays the onset of rebellion and maturity. The former youth may rebel against parental values and may seek support from people whom the parents would find repugnant. The latter youth may never break the "apron strings", remaining dependent until late in life.

To prevent a young person from experimenting with drugs (including alcohol and cigarettes) in a social context where experimentation is normative behavior is no easy task. Parents can convince a child to abstain until the child reaches middle adolescence, at which time the child may rebel against parental advice or rules. The parental behavior may provide an example to be followed (e.g., if the parent drinks alcohol excessively or

frequently takes tranquilizers, the child may smoke a lot of marijuana, thereby rebelling in choice of drug, but copying drug dependence). If parents subscribe to the notion that a child should abstain until adulthood, the child may long for the time when use is permitted, and may associate drinking or smoking with maturity. One danger is that secret experimentation with alcohol, drugs or tobacco may be unrealistically experienced as positive because of this association with maturity.

Clever advertising approaches to sell alcohol, other drugs and tobacco to the consumer is a major influence on consumption. They associate purchase and consumption of their product with meeting perceived needs (e.g., sexual beauty, maturity, wealth, power) of the target audience. Furthermore, the beverage alcohol and tobacco industries advertise to youth to prepare them to be good consumers (allegedly so they won't waste any time, once they reach legal age, until they buy the product) [B*Jacobson, Hacker & Atkins, 1983]. A recent study showing that the age of first sexual intercourse in adolescent girls was inversely correlated with level of cigarette smoking [J*Zabin, 1984] suggests that the advertising either selectively works in certain types of girls (perhaps those less closely and strictly supervised) or that the tobacco companies are also advertising sex. Recent ads also focus on the intrapsychic pain of troubled heavy drinkers (and perhaps troubled adolescents) with slogans like "get away from it all," "get it all together," and "everyone needs a little comfort" [B*Jacobson, Hacker & Atkins, 1983]. Since heavy drinkers purchase over half of the alcohol sold, keeping them drinking rather than seeking counseling is a sound marketing strategy for the industry.

Parodies on advertisements used to market these products are thought to be effective in inoculating young people against their influence [R*D.O.C.]. For example, teaching young consumers about the psychological tricks of advertising may fortify them against its influence to some degree. For example, having a group of teenagers make their own counter-ads is one way to help them fight the marketing techniques--they will always remember this project when they see an ad. The effectiveness of counter-advertising has been well demonstrated in the arena of smoking. Advertising of prescription drugs targeted at the physician probably influence prescribing practices more than physicians would like to admit. Methods to inoculate physicians against pharmaceutical propaganda have not been utilized widely.

Inoculation does not always work as intended. In one program, rehabilitated drug addicts warned adolescents about the dangers of drugs. But some students came away feeling safe to use drugs, confident that they could be rehabilitated. Some students found the stories of the addicts glamorous and exciting. Some students eventually purchased drugs from relapsing speakers.

Efforts to teach young people "responsible drinking" [B*Chafetz, 1982; J*Popham & Schmidt, 1978] have met with strong political resistance in the United States from the law enforcement community and from parents who adhere to the age prohibition

on drinking. Yet data from student surveys [B*Harford, 1984] suggest that according to self-reports, 85-90% of seventh graders have used alcohol at home and 47-54% drink frequently in this context. Furthermore, 65% drink in unsupervised contexts by this age. It is clear from these data and other surveys that by junior high school, it is too late to rely on refusal to experiment with alcohol. Drinking has become just as standard a part of the rites of passage of U.S. adolescents as necking and driving. Likewise, teaching drug users about drugs of abuse had a significant positive health impact during the late 1960's. While it did not appear to reduce street drug usage, it tended to decrease careless use. The development of cultural rules and rituals among the drug counter-culture may have decreased the number of drug-related visits to emergency clinics by youth and their families as they became more savvy in the "safe" utilization of drugs [B*Weil, 1972].

It is still a reasonable hypothesis that teaching teenagers, individually and as a group, to drink in moderation during adolescence might prevent common problems such as educational interference, unwanted pregnancies, drunk driving fatalities, and the development or progression of addictive disorders during adulthood. Wilkinson [B*1970] reviewed the literature on childhood experiences with drinking and points out that across several cultures the alcoholic respondents in retrospective studies reported, more often than controls, that they began to drink without parental support towards a moderate drinking pattern (i.e., they were either encouraged to abstain or to drink excessively). Furthermore, they tended to report ambivalence in the home towards alcohol, either between two parents or between parental teaching and parental example. This ambivalence may reflect knowledge among family members of the destructive capacity that alcohol has had on their family, and a desire to prevent the young child from setting off along that path. Yet the consequence of the emotionally charged nature of the message about alcohol seems, at least for some, to be an attraction rather than a warning. It may be hypothesized that the absence of parental support towards learning how to drink responsibly, analogous to support in learning how to walk, talk, drive, handle money, dress, etc., may lead the child to rely on external support, which often comes from other children who are experimenting with or addicted to alcohol, also drinking without reasonable parental guidance.

It is estimated that seven million (15% of all) U.S. children under age 18 live in, and twenty-two million adults come from, alcoholic homes (where at least one of the parents is alcoholic) [B*Woodside, 1982]. Other estimates, based on survey data, suggest that as many as 25% of U.S. residents are affected by alcohol problems in the home [B*Cahalan, Cisin, & Crossley, 1969; B*Gallup, 1980]. Similar estimates applied to other drugs of abuse are not available, but it has been estimated that one-third to one-half of abused or neglected children have a substance abusing parent [B*Black, 1982; B*Black, Mayer & MacDonald, 1978; B*Cork, 1969; B*Fitch et al., 1975; B*Kent et al., 1975]. Children of alcoholics or addicts (CoAs) are at high risk of al-

cohol or other drug dependence because of biological and learned factors. On the other extreme, about 5-10% of children come from alcohol abstaining homes (i.e., everyone in the home abstains from alcohol consumption). The other 70-75% come from homes in which alcohol plays a role but the parents are not alcoholic. Teaching children of alcoholics to abstain from drugs or alcohol may be one way to reduce their risk. However, delivering an abstinence recommendation to this subpopulation through nonselective means such as the school may generate conflict between child and drinking parent or between drinking families and the school. Furthermore, most adolescents may disregard an abstinence recommendation in view of personal experiences within the family and awareness of the dominant role of alcohol in current western culture. On the other hand, responsible drinking messages may prove to be damaging for young people who carry a genetic biological risk of alcoholism [J*Goodwin, 1979].

Adults, especially the elderly, are prime targets of licit drug marketing. It is somewhat harder to find opportunities to teach adults to take drugs with caution [B*Gaeta & Gaetano, 1977; B*Gaetano & Epstein, 1979]. However, adult education programs, employee assistance programs, parent-effectiveness training classes, child-birth preparation classes, health consciousness organizations, continuing professional education programs, and religious groups may offer appropriate opportunities to impact upon adults who are not in recovery or treatment programs. Some clinics and hospitals offer classes to patients, and continuing education programs are sometimes featured on public television. In clinical settings, focusing on family rituals and their relation to drinking and taking drugs could be a way to work towards change. Families wishing to avoid the chemical dependencies may be encouraged to alter their family rituals from those which permitted their ancestors to develop addictions [J*Wolin et al., 1979 & 1980]. Likewise, families which are changing their rituals towards toleration of uncontrolled drinking or drug use might be encouraged to give more thought to it given the risks.

TEACHING HINTS: Interpersonal Innoculation

In a seminar setting, have a group of students discuss the content of a one hour lecture they might deliver to adolescents about alcohol, drugs or smoking. Or have them discuss a ten-hour curriculum in the school's health education program devoted to chemical dependencies. How might the residents teach young adolescents to deal with peer pressure, parental influences, and advertising? Have the residents offer to talk about drug abuse in a local school, fraternity house, or dormitory using their own outline or suggested programs developed by others [B*Bureau of Narcotics & Dangerous Drugs]. Have residents, individually or in groups, offer their assistance to a local association, employer, union, school, or religious group which is planning a prevention project [A*Your health and alcohol].

In a seminar, have students brainstorm ways to impart psychoactive drug refusal skills and compatible attitudes to

adults in their practice or community, to doctors, nurses and other health care professionals, and to teachers and parents of school aged children.

Ask the students to select a product (e.g., cigarettes, liquor, tranquilizers). Then ask them to collect a few ads from the lay press or medical journals. Then, as a group, sort through the ads to look for subliminal and overt gimmicks intended to persuade potential customers. The students might first read excerpts from Key [B*1976] or Packard [B*1957]. Have the students sort the ads by strategy or have them design some counter-ads with guidance from [R*Doctors Ought to Care].

In their clinical practices, have the residents identify families in which there are one or more children at the age of risk of alcohol, tobacco and drug experimentation. Have them discuss these issues in a preventive way with the families. Recommend that the residents consider the developmental stage(s) of the child(ren) and family in helping the parents to deal with this problem. Have them report back to their preceptor, supervision group, or seminar the results of their clinical discussions. Prior to the clinical sessions, suggest readings [B*Liepman, 1983; B*McKeag & Perry, 1983; B*Rutledge, 1983].

EVALUATION: Interpersonal Innoculation

In the clinic setting, observe how the resident advises a worried parent about dealing with peer influences on his or her child in relation to psychoactive drugs? Does the resident explore parental substance ingestion? How comfortable is the resident with this line of exploration? How does the resident counsel the parents to deal with the conflict between their advice to their child and that which the child receives from peers? How well does the resident use knowledge of adolescent development in counseling the parent?

OFFERING ALTERNATIVES

LEARNING OBJECTIVES: Alternatives to Substance Use

The resident will understand the hypothesis that alternatives to substance use prevent the development of substance abuse in a population. The resident will be aware of alternatives programs which can be found in the local community and the missing programs which the community may need. The resident will be attentive to needs of this sort among his or her patient population and will demonstrate the freedom to prescribe alternatives other than drugs for patient ailments.

COMMENTS: Alternatives to Substance Use

There are a variety of etiologic hypotheses suggesting that psychoactive chemicals serve as self-medication for tension, depression, phobias, insomnia, and other disorders. They usually serve as temporary aids to suppress overwhelming feelings, but if they are used chronically, it is believed that these drugs lose effectiveness, and create additional problems of their own. Social theorists postulate that problems such as unemployment, poverty, broken homes, poor education, boredom, and neighborhood deterioration promote substance abuse. Critics of this view say the reverse is true.

Many people have remedies for most of their complaints which work well enough that they never consult the physician. Some of these remedies are commonly available psychoactive substances. When patients do consult physicians, there is a considerable risk that they will receive a prescription for a psychoactive drug, especially if the problem is in the psychosocial realm and the solution eludes the busy physician.

Prevention efforts following the alternatives model have addressed these issues by attempting to provide alternatives to mood altering drugs, such as community drop-in centers, cultural identity reinforcement programs, summer job programs, drug-free recreational facilities (e.g., un-bars, youth groups, camps), crisis counseling centers, foster-grandparents programs, relaxation training, among others.

In view of current uncertainty whether this strategy does help prevent substance abuse problems, resources are often not available to create alternatives programs. Nevertheless, family physicians may have opportunities to help support community groups which are working on such projects by contributing their name, leadership skills, financial contacts, and clinical knowledge or experience. At a clinical level, the physician may become proficient in prescribing non-drug alternatives which are likely to help resolve a patient's problems.

TEACHING HINTS: Alternatives to Substance Use

Have the student write a brief paper describing a strategy to decrease the prevalence of substance abuse by offering the population at risk alternatives. Direct the student to consider how this could be evaluated both in terms of appropriate utilization and effectiveness.

In a seminar setting, have the students survey the community in which their patient population lives, visiting a variety of alternative resources that they have identified. Have them describe these to one another so that by the end of the seminar, everyone knows what is available and what is needed in the community.

FAMILY MEDICINE CURRICULUM GUIDE TO SUBSTANCE ABUSE

In the office setting, have the residents use the prescription pad to prescribe non-drug alternatives to patients who presented with problems of a psychosocial nature. Have them save copies of their prescriptions to share with one another and their preceptors for a series of case presentations.

CASE 1: Mr. Jones was an 83 year old widower with congestive heart failure and anxious depression. His dog of twenty years had recently died. Since then he had trouble sleeping, fatigue, dyspnea, anorexia and had remained cooped up in his house allegedly because of ill health. He was noncompliant with his salt restriction and medications, a point which became evident through several brief hospitalizations. He was prescribed to purchase a new dog. The dog demanded daily brisk walks which demanded better cardiac function which produced better compliance and broke the loneliness and social isolation of the depression. The exercise improved his appetite and sleep naturally.

EVALUATION: Alternatives to Substance Use

How often does the resident prescribe non-drug alternatives to the patient with psychosocial problems? How creative is the resident in using these alternatives to explore ways to help patients to resolve their problems? Can the resident describe available alternatives programs in the community? How well does the resident know what they are like and how they can be best used?

HIGH-RISK COUNSELING

LEARNING OBJECTIVES: High-risk Counseling

The resident will be aware of the strengths and weaknesses of risk assessment techniques for the chemical dependencies and will be aware of recent progress that has been made in the biological and sociological study of risk of the addictive disorders. The resident will utilize tactful counseling techniques to warn patients and their families of impending danger of succumbing to the addictive disorders, and how to reduce their risks or to avoid increasing them. The resident will demonstrate skill in monitoring risk over extended time intervals and in providing constructive feedback to the patients and their families concerning efforts to reduce risk or avoid the development of chemical dependencies.

COMMENTS: High-risk Counseling

Counseling patients who are at high risk of chemical dependence or the harmful consequences of substance use is a selective

process which requires risk assessment first and then provision of counseling services once the target patients have been identified. Details about the identification of high risk individuals or families has been left to Chapter 3 of this monograph. Suffice it to say that risk factors include:

1. biological genetic tendencies,
2. other social and psychological factors related to presence of other substance abusers in the family,
3. prior personal history of an addiction,
4. cultural or ethnic background which has been associated with high prevalence of addictive disorders,
5. concurrent illnesses which impart greater than average vulnerability to the harmful effects of certain psychoactive substances used chronically or in high doses,
6. regular or frequent use of the abusable substances,
7. and attitudes which may decrease the person's ability to block progression of the disease process.

We may not be too far away from the day when we can test biologically for who is at high risk of the addictive disorders. Biological markers of risk of alcoholism have been found in children of alcoholics (who themselves have not yet begun to drink excessively or at all) which are also found in their alcoholic parents and which distinguish them from non-alcoholic families [B*Goodwin, 1984]. These findings promise to eventually provide clinical tools for identifying future alcoholics and may also lead to etiologic answers:

(1) The P_3 evoked brain potential [J*Porjesz & Begleiter, 1982] of alcoholic men and their non-alcoholic juvenile sons is distinct from that found in non-alcoholic controls and their juvenile sons. While the meaning of this finding to overall brain function is not understood, speculation has linked it with hyperactivity and learning disabilities, which seem to run in alcoholic families and alcoholics. It has been suggested that the altered P_3 potential reflects a biological difference in brain wiring which, in some unknown way, may predispose the alcoholic to drinking problems.

(2) Electroencephalographic studies of alcoholics demonstrate a lower percentage of alpha waves than is normally found [J*Propping, 1978]. Sons of alcoholics likewise have a shortage of alpha waves, but once ethanol is given, the distinction between EEGs of family history positive (FH+) and family history negative (FH-) men disappears. Specifically, in contrasting (FH+) young non-alcoholic men with matched FH- men, the study showed greater increases of slow alpha activity, greater decreases of fast alpha activity, and greater decreases in mean alpha activity in the FH+ group after low dose (0.5 gm/kg) alcohol ingestion [J*Pollock et al., 1983]. It has been hypothesized that this may reflect differences in brain aldehyde levels and that the resultant subjective experience is more reinforcing to the FH+ group. However, there appears to be a baseline difference which may be a wiring difference in the brain. In any case, alcohol seems to "normalize" the FH+ men.

(3) Serum acetaldehyde levels are higher following a drink in alcoholic men and their non-alcoholic young adult sons than in FH- men of similar age and drinking history [J*Schuckit & Raynes, 1979]. This finding is consistent with emerging evidence that racial differences in risk are biologically determined by ethanol and acetaldehyde metabolism [B*Zeiner & Paredes, 1978]. Oriental people who suffer an unpleasant flushing reaction after ingestion of ethanol (it resembles the disulfiram-ethanol reaction) have a faster elimination rate of ethanol and also a higher level of acetaldehyde during the ethanol elimination (presumably because the acetaldehyde elimination rate is slower than the production rate) [B*Deitrich & Collins, 1977; J*Ewing, Rouse, & Pellizzari, 1974; J*Farris & Jones, 1978; J*Hanna, 1978; J*Ijiri, 1974; J*Reed et al., 1976; J*Wolff, 1972 & 1973]. Native Americans in Reed's [J*1976] study had the highest acetaldehyde levels compared to Orientals and Caucasians, but other studies are inconsistent. This may be due to the use of different tribes of Native Americans in different studies; the prevalence of alcoholism also varies between tribes from very rare to very common [B*Zeiner & Paredes, 1978]. There is an hypothesis that acetaldehyde from ethanol catabolism condenses chemically with brain amines to form opioid-like tetrahydroisoquinolines (TIQs) [J*Cohen & Collins, 1970; J*Davis & Walsh, 1970]. Intraventricular (brain) or intrathecal injections of tiny amounts of one of these TIQs increased alcohol preference in several animal models, suggesting that racial or hereditary metabolic factors protect some from and put others at high risk of developing alcohol addiction if they drink alcohol [J*Myers & Melchior, 1977]. If this hypothesis is proven to be correct, it may lead to biological screening tests for alcoholism vulnerability.

(4) Low platelet monoamine oxidase (MAO) activity in alcoholic probands was associated with a high proportion of alcoholic relatives, while high platelet MAO activity was associated with few alcoholic relatives [J*Major & Murphy, 1978; J*Sullivan et al., 1978]. This suggests that an inherited form of alcoholism may be linked genetically with MAO activity.

The other drug dependencies are less well understood in terms of hereditary factors. However, drug dependencies seem to run in alcoholic and drug dependent families [B*Ziegler-Driscoll, 1979]. There is emerging evidence of endogenous brain compounds which bind to the same brain receptors to which benzodiazepines, sympathomimetic stimulants, and opioid exogenous agonists and antagonists bind. There are also hypotheses that opioid dependent individuals may be genetically predisposed to this condition by abnormalities in their brain endogenous opiates or their opiate receptors. As yet no evidence of this has been discovered.

Counseling

Once individuals have been identified to be at risk, they are entitled to some risk factor counseling. Specifically, they can be told of the addictive disorders and their higher risk. They can be exposed to opportunities to learn about the disorders

and to frequently assess their continuing risk. In cases where some of their risk factors are alterable, they may be counseled on how their risk can be reduced. Over time, these individuals can be monitored for changes in risk status just as one treats and monitors vascular hypertension.

CASE 2: Jack is a moderate drinking 21 year old married welder of Irish Catholic descent who comes from a family replete with alcoholic males. He has youth onset (type I) diabetes mellitus and grand mal epilepsy. He takes phenobarbital, phenytoin, and 58 units of lente insulin. Jack likes to drink beer after work with his friends from the job, and on weekends with his wife.

ADVICE: Jack can be counseled about his vulnerability to certain biological consequences of drinking (i.e., seizures whenever he suddenly reduces alcohol intake, drug interaction of his phenobarbital with ethanol in the liver and brain, difficulty in maintaining stable blood glucose levels with shifts in drinking). In addition, he should be encouraged to learn about alcoholism as it affects his family of origin. In that process, he will learn that he is at great risk of progression of his drinking to alcoholism, despite his desire to be a different kind of drinker than his father and uncles. He may also discover alcoholism in his wife's family of origin. He may begin to seriously consider abstaining or markedly reducing his drinking. In addition, he and his wife may begin to examine the role alcohol plays in their family rituals [B*van der Hart, 1978; J*Wolin, et al., 1979 & 1980] and may begin to alter its role in a constructive way. Eventually, Jack may request or accept alteration of his anticonvulsant to a non-sedative drug. And he may begin to develop ways of relaxing (e.g., exercise, yoga, meditation, hobbies) other than 3-4 beers before dinner.

This process of risk counseling is a part of health promotion and maintenance [B*Liepmann, 1983]. It is important to do this assessment process on a regular recurring basis just as one checks the heart, lungs and the cervix or for TB, syphilis or urinary tract disease. As noted in Chapter 3, the addictive disorders are among the most common categories of diseases and they are definitely among the most damaging. It is recommended that the resident develop a plan for assessment and risk counseling throughout the family life cycle. For example, it has been shown that very young children can name and recognize alcohol, and know, generally, its health and behavioral consequences [J*Jahoda & Cramond, 1972]. Yet it has unfortunately been rare for family physicians to discuss with parents the messages they are sending to their young children about alcohol, or for that matter, about caffeine, tobacco, or other drugs, until after one of the children has reached puberty and, in most cases, has begun to have substance abuse problems. It is wise to use the opportunities afforded by otitis media, upper respiratory infections, or camp physicals as a time to engage the parents in the process

of preventive health care including substance abuse risk assessment and education.

To increase efficiency and decrease the cost of this health promotion, one can offer "wellness" classes in which one addresses a variety of issues, (i.e., sexuality, puberty, menopause, discipline, dating, seat belts, motorcycles, drunk driving, retirement, placing elder family members in nursing homes, dealing with disabilities, the dangers of polypharmacy). Such classes may work best if topics are selected to be relevant to certain stages in the family life cycle to attract families in similar stages. As a side benefit, the cohort of families who attend the course together may develop supportive mutual relationships in the community. The substance abuse issues can and should be integrated within more general health education series rather than addressing them in separate courses. Some people will stay away from classes strictly on substance abuse because of fear of being stigmatized by their interest or worse, for fear that they will learn something which will force them to confront a substance abuse problem in their own family.

Another technique for efficiently delivering some of these messages to patients is to utilize otherwise wasted waiting room time to have them view videotapes or listen to audiotapes purchased or made by the family doctor which address common preventive issues. The family physician might prescribe a tape about normal sleep patterns, and how they change with aging, in order to convey dangers of sleeping pills and to debunk mythology about insomnia while a 42 year old patient awaits her urinalysis result. Or the physician might play a tape on adolescent drug and alcohol consumption [A*Cheers; A*Lisa; A*School days; A*Some of the things that go on out there] while doing a CBC on her 10 year old daughter. Then, after the patient has completed the tape(s), the physician can discuss the topic(s) further to provide a personal touch and to assess its impact on the patient without devoting a large block of time from a busy schedule. One can also prescribe a tape to be presented while awaiting the next office visit [A*A slight drinking problem; A*Alcohol: Crisis for the unborn; A*Alcohol: How much is too much?; A*Alcoholics Anonymous: An inside view; A*Angel Death; A*Another Chance; A*Born with a habit; A*Elder Ed; A*Feelin' no pain; A*Francesca Baby; A*Gale is dead; A*If you loved me; A*I'll quit tomorrow; A*Loosening the grip; A*Lots of kids like us; A*One day; A*Soft is the heart of the child; A*The Enabler; A*The new life of Sandra Blain; A*The secret love of Sandra Blain; A*Valium; A*Your health and alcohol]. There are pamphlets available on some of these issues, which may suffice as an inexpensive alternative to video or audiotapes.

TEACHING HINTS: High-risk counseling

In a seminar setting, have the students develop a schedule for when during the family life cycle certain preventive health promotion issues, including those related to substance abuse, should be addressed. Where students disagree with one another

about timing or topics, have them debate the issue using data from this monograph and other sources to defend their positions.

In a clinical setting, have the residents apply the schedule developed above to a sample of the families which come to them for care. Videotape or observe the content and process of their interactions to evaluate what was said and its appropriateness, how the messages were delivered, and the reaction of the family to the information or advice. Provide corrective and supplemental feedback to improve future attempts.

It is important to warn the residents that "scare tactics" are not to be considered synonymous with risk counseling. Patients may not be pleased to learn of their high risk of developing the dreaded addictive disorders. Just as "bad news" about a developing cancer is unpleasant, being informed of one's risk of becoming addicted is likely to induce defensive denial unless delivered very tactfully. The resident may need to experiment a bit, watch teacher-clinicians who are more skilled at delivering "bad news", and observe his or her own interactions with patients on videotape. Residents may wish to try out some approaches with one another in a role play situation before trying it with real patients. If substance abuse risk assessment and counseling is integrated into an overall process of health risk assessment and counseling, it may be more palatable for the patient, because there will be no feeling that one has been singled out to receive this "bad news". One may need to address the general issue of stigmatization of substance abuse in our society in order to reduce fear of stigma as a stimulus for denial. One may also need to preface comments supportively (e.g., "I am bringing this to your attention because I was worried when I realized that you are at much higher risk for alcoholism than the average patient in my practice because of several different factors in your background. Specifically I have identified the following factors which increase your risk..."). The resident should learn to offer suggestions of how the patient or family can lower risk or prevent unintentional elevation of risk in the future, while helping them accept and come to terms with those factors which cannot be altered (e.g., one cannot change one's heredity, ethnic background, or family history).

EVALUATION: High-risk counseling

During clinical care and precepting, observe the frequency with which the resident spontaneously introduces risk counseling about substance abuse issues. How effective is the resident in introducing the issue? How does the resident deal with patient resistance to discussing the issue? How well does the resident follow through on issues raised at prior visits? Does the resident clearly identify the risk factors and help the patient to cull those which are reversible from the others? Does the resident help the patient to discover ways to reduce risk or to prevent increasing risk? How well does the resident bring the family into the process of dealing with these issues? How well does the resident avoid overly simplistic scare tactics?

Also watch documentation in progress notes and the problem list to see whether substance abuse issues are given equal legitimacy to other strictly biological illnesses or risk factors. For example, if a resident is caring for a smoker, does the resident consider the tobacco use or dependence a diagnosis worth listing if: (1) there have not yet been any health consequences from the smoking, (2) the smoker is pregnant or living with someone who is [B*Meyer, 1983], (3) the smoker has small children or adolescents in the home, (4) the smoker has had at least one health consequence from the smoking, (5) the smoker has attempted to quit smoking or to seek assistance in quitting. If the problem list reflects only categories (4) and (5), it is unlikely that much effort is being spent on prevention of smoking by this resident. It would be considered gross negligence if a resident discovered vascular hypertension in a patient but never recorded it in the record, failed to place it in the problem list, and failed to treat or follow the blood pressure in subsequent visits. A similar standard needs to be set for substance abuse risk factors if excellent biopsychosocial care is to be achieved.

SECONDARY PREVENTION

LEARNING OBJECTIVES: Secondary Prevention

In the course of medical practice, the resident will be capable of recognizing early cases of chemical dependence or abuse among his or her patient population and then intervene quickly and decisively to prevent unnecessary progression of the illness to later stages.

COMMENTS: Secondary Prevention

Secondary prevention of chemical dependence involves identification of cases early in the course of the illness through screening and diagnostic attention. What is being prevented is unnecessary progression of the disease and consequential avoidable damage. Secondary prevention is most cost effective and efficient if limited to the high-risk population, but in terms of the human suffering involved, it is important to realize that no patient, despite the apparent low risk, is exempt or immune from becoming chemically dependent. The stereotypes of the typical heroin addict or the skid row alcoholic are far from universal. In fact, they are often so wrong as to be misleading. For instance, only about 5% of alcoholics live on skid row, and more than half of the skid row population abstain from alcoholic beverages. In medical practice, it is helpful to have regular screening procedures in place so as to not miss cases in their early stages. It is important for the family physician to be familiar with the natural history of the illnesses he or she is trying to detect and diagnose [B*Jellinek, 1960; B*Vaillant, 1983].

Some people consider family treatment of substance abusers to be a form of secondary prevention. The whole family is at high risk of developing drug problems. During family treatment of one substance abuser, others within the family may also be diagnosed and treated. In addition, recovery of one patient may precipitate the onset, exacerbation, or relapse of chemical dependence in another family member as a side effect of treatment. The careful surveillance of family members during family life cycle transition periods such as recovery from an illness, other external stresses (e.g., change of job, injury, death of a family member), natural milestones of life (e.g., starting a family, emptying of the nest, retirement) will reduce the delay in recognizing the initial phases of substance abuse problems. Examples of early warning signs might include increases in substance ingestion patterns or attempts to control ingestion, shifts in attitudes towards psychoactive chemicals in various family members, requests for drugs, reports of overwhelming stress or increasing family problems, and vague somatic complaints [Liepman, 1983].

The stages of the addictive disorders has not been uniformly agreed upon by experts. However, there is widespread consensus that the addictive disorders do progress gradually along a continuum of severity [B*Jellinek, 1960; B*Vaillant, 1983] if not arrested. Some models of staging utilize degree of resource loss (e.g., stage 1: no resource loss; stage 2: only reversible loss; stage 3: reversible and irreversible loss; see Chapter 3). Other models use medical or social parameters, dosage of intake, or duration of problems. Each model of staging has its advantages and disadvantages. For instance, while dosage of intake may be a good indicator of severity in the early phases of alcoholism (i.e., as the dosage rises, so does the problem severity), a decline during the later phases may not indicate less abuse, rather it may reflect the presence of severe damage to the liver (cirrhosis), hence the patient drinks less because the alcohol is removed more slowly.

It is often harder for clinicians to intervene with patients early in an illness because (1) patient and family denial has not yet been shattered by heavy resource loss, and (2) the diagnosis may not be obvious. It is important, though not often easy, for the primary care physician to maintain a conviction that it is necessary to intervene and that the diagnosis is accurate. Motivation to accept treatment and to discontinue the abused chemical may be more difficult in the early stages (see Chapter 4), but if initially unsuccessful, the family physician can tactfully persist in raising the issue as each and every consequence arises. This approach prevents the patient and family from incorporating their life stressors into the "reasons why I drink" list rather than the "reasons I should quit" list. It is important to realize that most people use psychoactive substances for good reasons, and that one who tries to take these substances away may be seen as hostile to the person. It is sometimes helpful to use the conception that we are attacking a vicious disease which is harming the nice patient rather than that the patient and the disease are inseparable (as is the inference when we call a patient "an addict" or "an alcoholic").

One very important area of secondary prevention is protection of children from the consequences of chemical dependency. Sullivan [J*1899] reported a mortality rate in children of alcoholic mothers 250% that of sober mothers of similar stock [B*Rosett, 1976]. 10% of children were killed by child abuse or neglect [J*Liepman, 1980; J*Sullivan, 1899]. Furthermore, children who survived have been described as: "morose and languid" [B*Burton, 1621, citing Aristotle; B*Rosette, 1976], "weak, feeble and distempered" [B*Morris, 1759; B*Rosett, 1976], and "diseased and puny in its corporeal part and beneath the standard of a rational being in its intellectual facilities" [B*Rosett, 1976; B*Trotter, 1813]. Undoubtedly referring to the Fetal Alcohol Syndrome, these authors were far ahead of their time, because the first reports in the scientific literature did not appear until 1968 [J*Jones & Smith, 1973; J*Lemoine et al., 1968; B*Ulleland, 1972]. Subsequent study has identified the Fetal Alcohol Syndrome (FAS) and the Probable Fetal Alcohol Effects (PFAE) conditions the third most common cause of mental retardation in the United States [B*Jones, Smith & Hanson, 1976]. Caused by the chronic presence of alcohol in the fetal circulation during pregnancy, the FAS can permanently damage the developing fetus producing mental and physical retardation and facial, limb and organ anatomical anomalies [B*Ouellette & Rosett, 1976; B*Rosett, Ouellette & Weiner, 1976]. Early studies demonstrated the complex interrelations between maternal drinking, smoking, nutrition, drug abuse [B*Meyer, 1983; B*Rosett, Ouellette & Weiner, 1976; J*Streissguth et al., 1980]. However, subsequent human and animal research has determined that these other factors are not the cause of the FAS. Nevertheless, drinking during pregnancy does not always result in abnormal offspring, however the dose response curve is definitely present [J*Ellis & Pick, 1980; B*Ouellette & Rosett, 1976; J*Sokol, Miller & Reed, 1980]. It is imperative that women of child-bearing age and their sexual partners be aware of the risks to the fetus of alcohol-induced teratological damage and growth impairment. Perhaps we need to modify a popular slogan: "If you drink, don't drive; if you drive, don't drink!" to warn women not to conceive and gestate while intoxicated. It is also important that they receive sensitive and informed clinical advice once it is too late to prevent the coincidence of conception and intoxication [J*Rosett et al., 1978].

TEACHING HINTS: Secondary Prevention

Ask the students to devise a strategy for sorting their patients into quartiles of risk for developing chemical dependence. Then ask the students to design a cost-effective plan to frequently check the patients at highest risk for the early warning signs of developing substance problems and to periodically check everyone in their practice. Show them a film on early diagnosis and intervention [A*Alcoholism and the physician, 1981; A*Alcoholism: Early Diagnosis and Management, 1981]. Have them suggest early warning signs, criteria for concern, clinical tests, time intervals, recording flow sheets, and a staging schema. In the clinic setting, ask residents to try their proce-

dures for a month on a randomly selected third of their patients, keeping track of the results. Compare notes among residents. Compare the prevalence rates among these high-risk patients in contrast to estimates in the literature. How accurate were the quartile risk estimates? How many newly diagnosed cases of alcoholism or other drug dependence were diagnosed in the clinic? Which are the most cost-effective screening methods? Have the residents select several early cases in which they will attempt to intervene to bring the chemical abuse problem into remission. These cases should be carefully supervised to assure that the zeal with which they attack the illness does not alienate the patient.

Have the students watch the film [A*Alcohol: Crisis for the Unborn] and then discuss the methods for preventing and/or dealing with the clinical consequences of drinking during pregnancy. In clinic, have the residents monitor alcohol, drug and tobacco intake in their pregnant patients and young parents of small children, being especially attentive to risk of child abuse or neglect in those families where there is a lot of drinking or drug use [J*Liepman, 1980].

EVALUATION: Secondary Prevention

How often do the residents check their high risk patients for substance problems? How do they check? How do they interpret the results? What do they do if it appears that a patient is progressing deeper into chemical dependence? At what stages do they find cases and at what stage do they intervene? Did the intervention approach alienate the patient? Was the intervention successful?

TERTIARY PREVENTION

LEARNING OBJECTIVES: Tertiary Prevention

The resident will be familiar with the treatment of consequences of the addictive disorders and will realize that these consequences are signs of an underlying addictive disorder. The resident will attempt to initiate successful rehabilitation with every request to treat the consequences until the underlying disorder has remitted. The resident will have a mechanism for dealing with burn-out and therapeutic nihilism in resistant cases.

COMMENTS: Tertiary Prevention

Tertiary prevention encompasses the prevention of consequences of chemical dependence. There are many consequences that can arise in the physical, psychological, and social arenas. For an extensive list of these, consult Table 3-1 or [B*Wartenberg & Liepman, 1984]. Clearly the best way to prevent the consequences of chemical dependence is by arresting the underlying illness through rehabilitation. However, physicians may also be in a

position to improve the health outcome in other ways which reduce the immediate damage irrespective of whether the patient accepts rehabilitation. The monograph will not address tertiary prevention at length because there is extensive and widely known information on the treatment of alcohol and drug related consequences [B*Pattison & Kaufman, 1982].

Of ethical importance is the physician who finds him or herself in the position of an enabler [J*Koppel, Stimmeler & Perone, 1980; J*Levinson & Ashenberg-Straussner, 1978] (see Chapter 4 or 8) whose actions are making it easier for the addicted patient to continue drinking or taking drugs without rehabilitation. For instance, the physician who prescribes antibiotics, theophylline and bronchodilators for a chain smoker with chronic bronchitis is helping the person to continue smoking until the emphysema has progressed further. With this kind help from the physician, the patient may not quit until a substantial irreversible disability has developed.

Aside from arresting the chemical dependence, there are other strategies for preventing complications. For instance, one may promote a decrease in drinking of an alcoholic, which may temporarily slow down the progression of the illness. One might give parenteral vitamin supplements to alcoholics in the hope of preventing Wernicke's encephalopathy, vitamin B₁₂ and folate deficiency, and other avitaminoses. Dosage reduction of smoking, drinking, and pill-taking during pregnancy may reduce the risk of malformed infants, though not as well as abstinence. Cimetidine treatment may reduce ulcers in drinking and smoking patients. Sedative hypnotics may temporarily improve sleep or nervous symptoms in alcoholics. Antidepressants may relieve depression in opiate or sedative dependent patients. Medically supported detoxifications may reduce the risk of seizures or delirium tremens in alcoholic patients. Stricter enforcement of drunk-driving statutes and better highway construction and auto safety engineering may prevent highway fatalities. Self-extinguishing cigarettes may reduce the prevalence of burn injuries. Gun control measures may reduce handgun homicides or suicides by chemically dependent persons. All these measures may be helpful in containing the damaging effects caused by untreated chemical dependence, but should not be construed as substitutes for treating the underlying illness.

There occasionally will be cases where the patient resists rehabilitation but still requests and requires medical care for the consequences of the unremitting addictive disorder. The resident may need assistance in developing a personal support system to help deal with "burn-out" and thoughts of therapeutic nihilism. Contact with Alanon may be helpful in some cases, because the physician often becomes frustrated as do family members. In addition, it may be helpful to increase the size of the team that is working with the resistant patient (i.e., include family members, some members of AA, substance abuse treatment professionals). Finally, it may be helpful to share ones frustrations at a Balint [B*1957] style group of peers.

TEACHING HINTS: Tertiary Prevention

Divide a group of students into two subgroups: the first should defend the paternalistic preventionists, those who would like to restrict the alcoholic or drug abuser from being able to make risky decisions (e.g., grounding an airline pilot who drinks, or forbidding a sufferer from emphysema to smoke); the second should defend individual freedom and victimless crimes. Debate the ethical, economic, and political issues. Should the community exercise coercion to accomplish preventative goals as is currently done with drunk drivers, psychopaths, and tuberculous patients? Have students role-play patients and the community at odds over a scenario of this sort, as in a courtroom setting. Should Jane Doe be allowed to continue to drink during her pregnancy? Should John Smith be allowed to smoke in the elevator he runs? Should Mary Jones be permitted to continue to drive her school bus under the influence of all her tranquilizers? What role will her doctor take when confronted with this ethical conflict of interest between the patient and the community (many of whom might be his or her family or patients).

In the clinical setting, help residents to recognize which of their clinical actions are tertiary prevention. When they apply a measure of tertiary prevention, ask them to explain what plans they have to intervene against the underlying addictive disease.

EVALUATION: Tertiary Prevention

Monitor the resident's cases of smokers and heavy drinkers over time to see whether the resident has been able to move beyond "band-aid" medicine in addressing the whole patient. Has the resident been able to overcome patient and family denial? Does the resident have a personal hangup about interfering with the patient's personal freedom? If so, the resident may need some counseling (see Chapter 9).

EPILOGUE

This chapter has attempted to introduce the family practice educator to the concepts and methods of prevention of substance abuse problems. It is hoped that this will encourage family physicians to take a leadership role in efforts to reduce the harm being caused to patients, their families and the community by substance abuse problems. Further information may be obtained by examining [B*ATPM Monograph; B*Miller & Nirenberg, 1984].

CHAPTER 8:
CHEMICAL DEPENDENCY AND THE FAMILY
By Raymond C. Anderson & Michael R. Liepman

OVERALL OBJECTIVE

The resident should be able to recognize the impact upon the entire family of chemical dependence in one family member. The resident should comprehend the association between one member's abuse of chemicals and behavioral, psychosomatic or psychosocial symptoms in other family members. The resident should understand how the family often interacts counterproductively with the chemically dependent person and how the family can correct this. The resident should be capable of utilizing the family in all phases of diagnosis, motivation, treatment and follow-up care. In addition, the resident should recognize the positive features of relapse and the negative consequences of recovery of the family and know how to therapeutically intervene.

COMMENTS

Chemical dependency has a profound impact on the family and other people associated with the family. The addicted family member's use of chemicals, and his or her resulting behavior, has a profound influence on virtually all members of the family and on anyone else who has an emotional bond with the addicted person. Conversely, family members and other members of the social support network have a profound, rarely constructive, influence on the behavior and attitudes of the person who suffers from chronic chemical dependence [B*Johnson, 1973; B*Liepman, Williams & Whitfield, 1984; B*Liepman, Wolper & Varquez, 1982; J*Koppel, Stimmler & Perone, 1980]. For simplicity, the term **CHEMICALLY DEPENDENT (CD) FAMILIES** will be used to designate families which have at least one member who suffers from chemical dependency.

Alcoholism runs in families. The vulnerability to catching alcoholism is probably partly inherited biologically [B*Goodwin, 1976; B*Murray & Stabenau, 1982; J*Schuckit & Raynes 1979]. Cultural, ethnic and family rituals may facilitate assortative mating among alcoholic families and may teach persons to use alcohol or drugs abusively [B*Cahalan, Cisin & Crossley, 1969; J*Wolin, Bennett & Noonan, 1979; J*Wolin, Bennett, Noonan & Teitelbaum, 1980]. Childhood roles in alcoholic families have also been associated with chemical dependence in adulthood [B*Ackerman, 1978; B*Black, 1982; B*Deutsch, 1982; B*Wegscheider, 1981]. There is a clinical need for genetic counseling in alcoholic families. In addition, risk assessment may provide an opportunity for early intervention and prevention of unnecessary progression of an existing case or onset of a new one.

LEARNING OBJECTIVE: Recognizing CD families

It is crucial that the family physician learn to recognize people whose problems stem from the use of alcohol or drugs, because such patients often fail to recognize that chemical use itself is at the root of their problems. Similarly, the family physician is responsible for identifying illness which is indirectly associated with chemical dependence in other family members. (For more details, see Chapter 3.)

COMMENTS: Recognizing CD families

Frequent clinic attendance, leaving the hospital against medical advice, multiple family problems, psychosomatic illness, frequently cancelled appointments or no shows, non-compliance, complaints of vague symptoms, non-response to conventional treatments, child abuse or neglect, absenteeism from work or school, and drug seeking behavior are all signs commonly associated with members of chemically dependent families. Because of social stigma and the desire for self-respect, patients often spare their physicians and themselves the embarrassment of confronting the chemical dependence directly by disguising the illness and associated behaviors as something else. In contrast to the malingerer, who consciously wishes to confuse the physician, members of chemically dependent families who suffer from denial fool the physician only indirectly as a consequence of fooling themselves. Uncovering this ruse begins the process of alleviating denial and facilitates facing the truth. The clues are often readily apparent to physicians who keep this diagnostic entity in mind with a high index of suspicion and follow up on unexplained irregularities.

TEACHING HINTS: Recognizing CD families

In a seminar setting, have the residents read books or articles about the families of chemically dependent persons [B*Ackerman, 1978; B*Black, 1982; B*Black, Mayer & MacDonald, 1978; B*Brooks, 1981; B*Cork, 1969; B*Deutsch, 1982; J*El-Guebaly & Offord, 1977 & 1979; B*Hornik, 1974; J*Jacob, Dunn & Leonard, 1983; J*Kaufman, 1980; B*Kaufman & Kaufmann, 1979; B*Liepman, 1984; E*Liepman, Williams & Whitfield, in press; B*Liepman, Wolper & Vasquez, 1982; J*Miller, 1980; J*Moos & Billings, 1982; J*Stanton et al., 1978; J*Steinglass, 1981; J*Triplatt, 1978; B*Wegscheider, 1981; B*Woititz, 1978; E*Woodside, 1982; E*Ziegler-Driscoll, 1979]. Then, have them improvise a skit about how such members of a family would be likely to present to the doctor. It might be helpful to have present some members of recovering families (preferably families with long-standing recovery through treatment and/or the self-help community) to coach the actor-residents, to comment on the authenticity of the roles, or to play roles in the skit. The residents could also role play their responses, as physicians, to these different scenarios, making an effort to focus at least partly upon the relationship of the chemical dependence to the presenting complaint.

Have the residents apply the Family AFGAR test to the next 30 consecutive patients that they see in the clinic [J*Good et al., 1979; J*Smilkstein, 1978]. For those families with high scores, have the residents do a thorough alcohol-drug pharmacological history to determine how many have chemical dependencies.

It may be helpful for the residents to be familiar with Smilkstein's [J*1980] concept of how families respond to stress and seek help from external resources. The residents could present two kinds of cases in order to contrast motivational states according to this model:

Case 1: A patient presents to the clinic complaining that she cannot sleep and needs some tranquilizers because she is going through marital difficulties. On inquiry, she reports that her husband has been staying out very late at night coming home intoxicated. She thinks he may be having an affair, because he has not approached her sexually for months, which is quite a change for them. She is interested in getting some help for the marital problems, once asked.

Case 2: A patient presents to the clinic for an insurance physical. He is nervous and has the odor of alcohol on his breath. Upon inquiry, he reports that he had "a couple of beers" with lunch. Physical exam shows hepatomegaly with tenderness, and enzyme elevation is found characteristic of early alcoholic liver disease. Upon further inquiry about drinking, he becomes defensively vague and pert. He states that he drinks no more than anyone else, and would appreciate it if the doctor stuck to his medical evaluation.

The two cases above could be replaced with real cases in one's practice in which one involves a family in disequilibrium and the other in pathological equilibrium.

EVALUATION: Recognizing CD families

The preceptor can assess the level of resident index of suspicion by asking the proportion of patients or families under their care that suffer from chemical dependence. If the estimates are very low (see Chapter 3), then it is likely that the residents are being fooled by patient denial most of the time, or they are not screening families for CD. The residents could be asked to identify and present several typical cases of chemically dependent families from among their caseloads, to determine whether they identified these cases themselves and whether the selected cases represent a full spectrum of stages rather than only late stages. The preceptor might ask the resident to see a family known (by the preceptor) to have chemical dependence. If the patient retains denial, the patient visit could be used as a test of the skill with which the resident penetrates denial. The

CHAPTER 8: CHEMICAL DEPENDENCY AND THE FAMILY

clinical debriefing could assess the index of suspicion that the resident assigns to chemical dependence in this family. If the selected family no longer has denial, then they could be forewarned, for teaching purposes, to refrain from spontaneously reporting the chemical dependence until asked about it skillfully by the resident. A series of clinical skills evaluation packages are available [R*Corley & Fisher] for family practice residents which use patients and actors. Several of these include the problem of alcoholism in the programmed patient.

LEARNING OBJECTIVE: Risk Assessment & Prevention

The resident should be able to identify persons who are at high risk of membership in a chemically dependent family. Furthermore, the resident should be capable of diplomatically inquiring about clues to CD in order to assess risk. The resident should demonstrate skill in counseling the patient and family about the best ways to prevent progression of an existing case or development of new ones. (For more details, see Chapter 7.)

COMMENTS: Risk Assessment & Prevention

The risk of being a member of a chemically dependent family depends upon a variety of factors including ethnicity, urbanicity, religious preference, stress, education, social class, race, location, etc. [B*Cahalan, Cisin & Crossley, 1969; B*Liepman, 1983]. While no factors absolutely predict nor conversely protect from CD, these risk factors contribute to the probability of CD in populations. From these data one can calculate risk probabilities for individual families or persons.

In U.S. families, there is a large degree of ethnic heterogeneity. As a result, attitudes and values concerning use and abuse of chemicals may vary even within a family. In such cases, unresolved interpersonal conflicts centering around drug use or abuse may create permanent schisms between spouses, siblings, or across generations. These conflicts may retard prompt action in recognizing and seeking treatment for CD when it reaches problematic proportions. These schisms may facilitate unrestrained drug experimentation by the youth in the family (e.g., Billy's father's untreated alcoholism blocks his criticism of his 10 year old son's experimentation with alcohol).

TEACHING HINTS: Risk Assessment & Prevention

The residents could role-play a family where one side has ethnic background which promotes heavy drinking in males (e.g., Irish) and the other side has background which discourages heavy drinking (e.g., Italian). The roles should include three generations (i.e., both sets of in-laws, husband, wife, and teen-aged children). Or a real family like this from the practice could be brought in for interview. In either case, questions about cultural norms and family rituals [J*Wolin, et al., 1979 & 1980] as

they pertain to alcohol or other drugs, abuse of chemicals, and recovery from addictions, may be quite revealing. If role play is selected, it is important for the role players to have previously researched the cultural groups they are representing. The discussion should include the different reactions that this family is likely to communicate to their drinking teenagers, how the alcohol becomes a pivotal family issue, and how drinking or drunkenness becomes symbolic of power in the family.

In providing preventive health care to these families, the risk of developing new cases of CD should be openly discussed with the family. As adolescents enter the world of drug pushers, drug and drinking parties, doctors who prescribe psychoactive drugs with little caution, bars, liquor stores, and compelling tobacco and alcohol advertizing, how will they choose to respond? How will their role models in life lead them to their choices? How will their parents counsel them, if they do? How will the adolescents respond to parental advice on drugs?

EVALUATION: Risk Assessment & Prevention

In a family suffering from CD being followed by the resident, evaluate the awareness of the cultural-ethnic determinants of the substance abuse behavior. One can ask: Has the resident discovered the conflicts over CD between different branches of the family? How does the resident use this information in motivating treatment for those who suffer from CD and in preventing new cases in the adolescents of this family?

FAMILY ROLES

LEARNING OBJECTIVES: CD family roles

The resident should learn to recognize the different family roles that develop in CD families. This knowledge should be usable in recognizing CD families by a role played by any family member. The resident should also be capable of assessing, counseling or referring a patient whose role in the CD family causes distress of an emotional or physical (e.g., the asthmatic child) or social (e.g., the juvenile delinquent) nature.

COMMENTS: CD family roles

Roles of members of CD families have been described in great detail [B*Black, 1982; B*Wegscheider, 1981]. The roles are described fairly distinctly, but there is considerable variation in them in real families. The roles often last a lifetime, but persons within the families can switch roles, or they may alter their roles over the family life cycle or with treatment. In most families, one can see the roles repeating themselves over the generations. The roles will be summarized briefly below.

The CHIEF ENABLER

In virtually every chemically dependent person's life, there is someone who has made the destructive use of alcohol or drugs easier. The CHIEF ENABLER is the person who is most involved in the enabling process. This role is generally filled by the spouse or parent of the chemically dependent person, but children, close friends, or work associates may also occasionally fill this role. In most cases there are lots of other "assistant enablers" who also participate in this process. However, their participation is usually more chaotic than coordinated and there tends to be lots of mutual undermining of one other. Motivated unwittingly by the benevolent desire to "help" the chemically dependent person to control the ingestion of chemicals and to avoid the damaging consequences of drug abuse, the enablers deny that a problem exists, accept responsibility for the drug abuse, attempt to conceal from others the behavior and its consequences, and become involved in the self-administration and/or supply behaviors ostensibly to reduce or modify the ingestion of chemicals. Despite the degree to which alcohol or other drug problems interfere with daily life and consume energy and effort, many CD families avoid verbal reference [B*Brooks, 1981] to this problem, alluding to it only indirectly (e.g., "Is she that way again?", "He's not feeling well today" or "There's lots of pressure at school right now"). Rather than call it alcoholism or other drug dependence, the CHIEF ENABLER usually leads the whitewash of the family reality, sometimes harshly suppressing efforts by others (e.g., children, relatives, friends) to surface their concerns. By assuming responsibilities that belong to the chemically dependent person, the enablers allow the destructive drug-taking behavior to continue far longer than it would have been endured without assistance and support. For instance, parents who recurrently pay for their child's damages done while under the influence of alcohol or other drugs, or a wife who repeatedly makes alibis to the boss about the reason for her husband's absence from work, is enabling the natural negative consequences of the abuse of chemicals to escape restraint, and hence supports the continued substance abuse by interfering with problem identification (i.e., the child or husband could say after the event: "I have no problems from my drinking or other drug use!"). Often the enablers will try to appeal to the chemically dependent person to stop or reduce the drug ingestion in order to reduce the negative consequences (which they have naively been suffering in proxy for the substance abuser). However, usually these attempts are ineffective because:

1. the enabler confronts the user in a hostile, punitive way such that the confrontation has little chance of success (e.g., "You are just a drunken bum. You only care about yourself."),
2. the enabler avoids confronting the disease and suggests that he/she will settle for changes far short of total recovery (e.g., "Why don't you cut down on your drinking for awhile?" rather than "You must enter a rehabilitation program for your chemical dependence!").

When these attempts fail, the CHIEF ENABLER will often try more aggressive maneuvers such as counting drinks, pouring out or watering down the liquor, avoiding situations in which problematic use usually occurs, restricting the person's access to family funds, physical violence, or nagging about the alcohol or drug ingestion. As each strategy fails, the enabler may attempt to deal with the problem by joining in the use or abuse of alcohol or drugs with the chemically dependent person, or by becoming invested in relationships outside of the family. The enabling behavior may stop when it is no longer possible to deny the problem, and the enabler leaves the family permanently. However, the enabler usually finds someone else with a similar problem who needs an enabler and the chemically dependent person usually finds a new CHIEF ENABLER, hence spreading the disease rather than curing it.

Throughout this process, the CHIEF ENABLER paradoxically feels both guilt ridden and helpless. The CHIEF ENABLER is often perceived by self and others as the "cause" of the chemical dependency behavior. The chemically dependent person may angrily blame the CHIEF ENABLER for attempting to control the ingestion behavior (e.g., "I drink because you nag at me so much!"). On the other hand, other family members may blame the CHIEF ENABLER for making it so easy for the CD person to continue. Usually attempts at controlling ingestion are ineffective except at stimulating defensive reactions in the CD person (e.g., hiding bottles, sneaking drinks or other drugs, lying, making excuses to justify ingestion, etc.).

The physician who identifies the enablers can play a significant role in guiding the whole family towards treatment and recovery, either as a whole or piecemeal as family members begin to accept the need for change. If even one family member begins to recover, this person can be provided support in carrying the message to others in the family, and eventually this process can bring the entire family into treatment (see Chapter 4).

CHILDREN'S ROLES IN ALCOHOLIC FAMILIES

Children in alcoholic families are also caught up in and harmed by the behavioral and social associations to chemical dependency. Children in CD families acquire roles [B*Black, 1982; B*Wegscheider, 1981] based on gender, birth order and spacing intervals, special talents or dispositions, major family stressful events (e.g., a divorce, death of a family member, a geographic move) and projective identification (i.e., they are "programmed" to behave in particular ways by others in the family to fill family voids). The roles are somewhat dependent upon the number of children in the family. In smaller families, a child may alternate roles or assume more than one role simultaneously. Over time, children may switch roles with one another, especially when a child leaves the home or begins psychotherapy.

The Role of the FAMILY HERO

The eldest child most often has the role of the FAMILY HERO. The HERO is helpful inside the family, and is an achiever outside the family. The HERO appears to be successful and well adjusted, but often suffers from internal emotional turmoil (e.g., depression, anxiety). The family HERO may be difficult to engage in personal treatment because of his or her external success. The HERO's external behaviors supplement family deficiencies secondary to the chemical dependency (e.g., the HERO at the age of seven may prepare dinner every night because mother is drunk in bed all afternoon, the HERO at age 10 may drive father home from the bar, the HERO may care for smaller children in the house much as a parent would do, forgoing the usual sibling rivalry behavior common during normal childhood).

Later in life FAMILY HEROES tend to be prone to ulcer disease, migraines, and heart disease. They are often Type A workaholics. They often choose motherhood or the helping professions as careers, such as medicine, nursing, missionary or social work. They often choose a mate who is or later becomes chemically dependent. Their selflessness gets them into trouble with emotional depression and close relationships (i.e., they tend to neglect their own needs and become very intrusive and controlling towards others). Low self-esteem and the secondary tendency to judge oneself harshly by the accomplishments and failings of one's children or spouse are prime prerequisites for the job of CHIEF ENABLER. The adult who grew from a FAMILY HERO is prone to burning oneself out by getting overcommitted to hopeless rescue missions.

The Role of the SCAPEGOAT

SCAPEGOAT children find themselves unable to cope with the FAMILY HEROES. Usually younger than the HERO, the SCAPEGOAT cannot compete with the external or internal competence of the HERO. With most attention coming from the HERO rather than the parents (who are usually too busy being chemically dependent or CHIEF ENABLER), the SCAPEGOAT seeks support outside the family's social fabric. They hide or run away, act out, begin to use drugs and alcohol, have problems with school or the law, fall into promiscuity and unplanned pregnancy, or involve themselves in problematic situations, especially during stress. The SCAPEGOATS bring disgrace to themselves and their families. They also bring the attention of helping professionals to their family if their behavior is perceived as a "cry for help" [J*Smilkstein, 1980]. They sometimes suffer from depression and enuresis. If the SCAPEGOAT has a chronic illness such as asthma, diabetes, or hemophilia, self-induced relapses of the illness may become a vehicle for acting out anger and for gaining power in the family. Teen pregnancy and suicide attempts may also serve a similar function. The SCAPEGOAT may be the first member of the family that a physician sees, perhaps because of the SCAPEGOAT's behavioral problems. This should serve as a diagnostic clue for the physician, and it may be the key to involving the entire family in an intervention. If the SCAPEGOAT is perceived as a bad or

defective child without considering the family context, the child may become permanently branded, and the family may continue to evade recovery by sacrificing the SCAPEGOAT.

In adulthood, the SCAPEGOAT may continue to be constantly in trouble. The end result of that may be prison or frequent psychiatric hospitalization. The diagnosis of sociopathy is frequently placed on such persons because of their lifelong struggle of oppositional behavior against authority, perhaps in the quest for attracting the parental attention that was sparse during childhood.

The Role of the LOST CHILD

The LOST CHILD is usually withdrawn and isolated. The LOST CHILD makes few demands on the family and attracts very little support or nurturance. A sense of self-worth is not developed. The LOST CHILD assumes responsibility and carries guilt for causing the family's problems. The LOST CHILD may invest time and energy in imaginary friends, or focus attention on a family pet or a plaything. Eating may provide gratification, and obesity may result. Later short-term pleasure may be sought in alcohol or drugs. Intimacy and interaction with humans is difficult, and decision-making is a problem. The LOST CHILD shares some similarities with the SCAPEGOAT, but the withdrawal is passive, in contrast to the aggressive behavior of the SCAPEGOAT. Depression, school avoidance, bed-wetting, allergies, asthma, and frequent injuries may be seen in the LOST CHILD, perhaps a feeble attempt to attract attention and nurturance. School performance may suffer as the child develops neither socially nor cognitively.

If the LOST CHILD comes into adulthood without having had any treatment, he or she may be unable to find gratification in personal relationships, may exhibit sexual role confusion, and may attempt to gain fulfillment through the accumulation of possessions. Obesity or the development of chemical dependence may occur. Marriage, if attempted, will be impaired by intimacy difficulties and the preference for withdrawal during stress. The reluctance to trust humans may interfere with psychotherapy as well.

The Role of the MASCOT

The MASCOT is usually the last-born child. Everyone in the family has already assumed a role for survival. The last child is protected from the "bad" things that happen in the family. The MASCOT finds that he or she can relieve the tension in the family by clowning. Laughter may temporarily relieve the family's anxiety. Because of the child's extroverted behavior, he or she is hard to ignore. Such a child figets, clowns inappropriately, knocks things over, and can appear hyperactive. It is important, however, to take a careful look at the family and the MASCOT role before chemical treatment is started for the child's hyperactivity.

Without appropriate treatment, the child's pursuit of attention will continue into adulthood. The child may be hypersensitive to stress, may feign physical illness, or become delusional and be diagnosed as schizophrenic. The child is also at high risk to become suicidal or chemically dependent. The adult MASCOT may be unable to resolve personal conflicts because of the tendency to laugh off stress and problems (i.e., escapism) and may resort to drinking whenever there is a problem to solve.

TEACHING HINTS: CD family roles

Have the residents view the movie about family roles or read about them [B*Black; B*Wegscheider]. Then have them view a movie showing CD families [A*Drink, Drank, Drunk (You Waste Your Life sequence); A*Family Secret; A*If you loved me; A*I'll Quit Tomorrow; A*Our Brother's Keeper; A*Soft is the heart of the child; A*The Enabler] and have them identify and describe the roles of the various members.

Interview a real CD family (recovering). Make a genogram for the family and have residents identify the roles of each family member through the generations.

Have the residents discuss how roles are assigned and maintained in families and how the chemical dependency alters this normal process in CD families.

EVALUATION: CD family roles

The resident could be quizzed to determine knowledge of the CD family roles. Questions could include:

QUESTION 1: Name and describe the roles various members of chemically dependent families enact.

ANSWER: The CHIEF ENABLER is a close and powerful enabler who tries to control ingestion and block negative consequences of substance abuse. This person usually feels helpless and guilty.

The FAMILY HERO is usually a very competent adult-like child, usually the first-born, who rescues the other family members and forgoes his or her own childhood in the process. This child is a master manipulator, using personal resources to whitewash the family troubles. This child becomes a prime candidate for a CHIEF ENABLER role in adulthood, and is also prone to stress disorders.

The SCAPEGOAT is often second-born and is the prime (non-adult) trouble-maker in the family. This child takes the spotlight off the trouble of the chemically dependent parent and obtains attention from within and outside the family. This child often brings the family into treatment via this trouble. Often this child is

diagnosed "hyperactive" or "juvenile delinquent". This child is very manipulative, using trouble as a tool.

The LOST CHILD is very withdrawn, having trouble making and trusting human attachments and relationships, hence a "loner". This child is prone to psychosomatic problems or schizophrenia-like emotional problems (e.g., schizoid or borderline personality).

The MASCOT is often the last-born child, being the family clown, who lightens up the family through humor and frivolity. This child may be diagnosed as "hyperactive". This child may find it difficult to be serious as an adult, and may laugh off problems rather than solve them.

QUESTION 2: Which CD family roles are associated with the childhood diagnosis of "hyperactivity"?

ANSWER: The MASCOT and the SCAPEGOAT.

QUESTION 3: Is there a role in the CD family which is not harmful in some way to the developing child?

ANSWER: No. The FAMILY HERO usually looks pretty good as a child if one looks only superficially at adult-like functions. But this child lacks spontaneity and the ability to relax and play. As an adult, this person will be a prime candidate for burnout having continuously committed oneself to hopeless challenges and usually being overextended.

QUESTION 4: Which CD family role(s) is/are associated with conflict resolution avoidance?

ANSWER: All of the CD family roles are prone to avoiding conflict resolution. The CHIEF ENABLER leads the way by denial and by trying to whitewash reality, with the FAMILY HERO following suit. The SCAPEGOAT and MASCOT tend to distract the family, while the LOST CHILD just withdraws. The chemically dependent parent tends to escape into chemicals when conflict arises.

QUESTION 5: Name some presenting signs and symptoms which might alert the family physician to role problems in CD families. Identify which roles are most likely to be associated with each.

ANSWER: Stress disorders such as hypertension, peptic ulcers, and heart attacks seem to be most often associated with the FAMILY HERO role. Psychosomatic disorders such as asthma, allergies, and enuresis are often associated with the LOST CHILD. Injuries often predominate in the MASCOT or the SCAPEGOAT.

QUESTION 6: Which childhood role is associated with problems in school?

ANSWER: The SCAPEGOAT, the LOST CHILD, and the MASCOT. The SCAPEGOAT gets into trouble with the school authorities, the LOST CHILD stays too withdrawn to learn real well, and the MASCOT is the class clown and tends to have trouble attending to serious matters.

Assess the residents' utilization and understanding of family roles by inquiring about family roles of members of CD families they are treating. When a case is presented which sounds like an offspring of an alcoholic (e.g., psychosomatic illness, relationship problem, gender identity problem, depression, etc.), inquire about the parental history of chemical ingestion to determine whether the resident is using knowledge of the roles to find CD families. If a case sounds like it may be a spouse of an alcoholic (e.g., depression, anxiety, insomnia, parent-child problems, marital problems, sexual problems, headaches, hypochondriasis, psychosomatic problems), check to see whether a family history of substance use has been obtained. Examine the family genograms on records of patients being cared for by the resident to see if the family substance abuse history has been recorded.

HEREDITY

LEARNING OBJECTIVE: Heredity

The resident will be aware of the hereditary contributions to the risk of developing or "catching" chemical dependence. The resident will be capable of explaining this to patients and their families so as to warn them about the risks of transmitting CD to future generations. The resident will be able to distinguish biohereditary influences from social influences on intergenerational transmission.

COMMENTS: Heredity

Alcoholism and other drug dependencies seem to run in families [J*Cotton, 1979]. At least for alcoholism, there is compelling evidence supportive of biological transmission of risk of alcoholism. For example, identical twins are twice as concordant for alcoholism as fraternal twins [B*Gurling et al., 1981; B*Kaij, 1960], and adopted sons of alcoholic parents are four times as likely to "catch" alcoholism as adopted sons of non-alcoholic parents [J*Bohman, 1978; J*Cadoret & Cath, 1978; B*Goodwin, 1976; J*Goodwin et al., 1973, 1974 & 1977; B*Murray & Stabenau, 1982]. Environment seems to play a more significant role for daughters of alcoholics than for sons. The drinking patterns of adopting parents seems to influence drinking patterns and the incidence of secondary alcoholism in adopted sons but not

upon the incidence of primary alcoholism, where heredity seems to be most important.

Hyperactivity of childhood (more recently dubbed the Attention Deficit Disorders or ADD) seems to run in the same families as alcoholism. It has been proposed that ADD is a consequence of being a child in the disruptive environment of an alcoholic family. However, there is evidence that children of alcoholics have different brain wave patterns (P_3 evoked brain potential) [J*Porjesz & Begleiter, 1982] and alpha-wave response to ethanol [J*Pollock et al., 1983; J*Propping, 1978]. (For more detail, see page 7-20.)

A number of different biological pharmacogenetic factors are being studied to determine whether they contribute to the hereditary risk of "catching" or resisting alcoholism. A high proportion of orientals suffer from an acetaldehyde toxicity reaction (i.e., flushing, nausea, lightheadedness, palpitations) when they drink only a small amount of ethanol. This is also seen in people of other races. It resembles the alcohol-disulfiram reaction and suggests a similar etiology (i.e., perhaps the acetaldehyde catabolism is relatively slower in these people than in normal drinkers). Persons with this constitutional factor tend to avoid alcohol. A less extreme form of this condition has been noted in alcoholics and sons of alcoholics who are not yet alcoholic [J*Schuckit & Raynes, 1979]. It has been proposed that there is insufficient acetaldehyde build-up to discourage drinking in this group, but that the excess acetaldehyde is available for sufficient time in brain tissue to permit chemical condensation reactions with dopamine to produce an opioid-congener called tetrahydropapaveroline (THP) which appears to stimulate alcohol self-administration behavior (see Chapter 1). This hypothesis would explain why certain persons sustain a greater risk of "catching" alcoholism than others, and why they may not develop it until they begin to drink heavily. It might also explain why so many people have the tendency to relapse later after a period of abstinence if they begin to drink again, even in moderation.

Environmental influences have recently been studied in terms of family rituals (i.e., recurring family gatherings at which family members participate in stereotypic behavior, such as family holiday gatherings, rites of passage, and family vacations). The integration of heavy drinking or drunkenness into these and other family rituals seems to perpetuate alcoholism throughout subsequent generations by birth or marriage [J*Wolin et al., 1979 & 1980].

Genetic studies of other drug dependencies have not appeared in the literature. It is clinically apparent that alcoholic families often have other drug dependencies as well. But, studies of hereditary transmission of drug dependencies have not been done. The high prevalence of alcoholism in methadone maintenance patients may be explained by the THP hypothesis that drinking in alcoholism may produce endogenous opioids in the brain which in turn might satisfy a craving for opioids. The biochemistry of other drug dependencies are currently a mystery and have eluded

explanation in this way. From epidemiologic studies on individuals, we only know that those who use other drugs excessively often do the same with alcohol.

TEACHING HINTS: Heredity

In a seminar setting, the residents might review the literature on hereditary [B*Goodwin, 1976; B*Murray & Stabenau, 1982] and social [J*Schuls & Wilson, 1973; J*Schwartzman, 1975; J*Seldin, 1972; J*Wolin et al., 1979 & 1980] influences on intergenerational transmission of alcoholism. The residents can present to one another the evidence that exists pertaining to each model of transmission. A debate between two groups of residents could pit biological inheritance against environmental influences.

In a clinical setting, the preceptor could review a sample of family genograms of patients being cared for by the resident. When a case of alcoholism or drug dependence is found in the family, the preceptor could quiz the resident about what had been discussed with the patient and family about the risk of developing chemical dependence. Videotaping of clinical visits by persons from chemically dependent families could provide the preceptor with opportunities to shape the resident's method of counseling such patients. In the setting of prenatal or premarital counseling, the resident could be encouraged to inquire about the alcohol and other drug histories of each branch of the family, so as to obtain information that might be useful in genetic counseling.

EVALUATION: Heredity

Examine the genograms in the clinical records to see if the resident is gathering genetic information about alcoholism as a matter of routine. In a case with positive family history, inquire about the implications this might have for the patient in terms of future risk. Try to determine what extra measures the resident might take to prevent the patient or other family members from "catching" alcoholism. Observe the resident counseling a patient about the hereditary and other risk factors he or she has.

FAMILY HISTORY

LEARNING OBJECTIVES: Gathering Family History

The resident should be capable of gathering historical information about chemical usage and its consequences from families or individuals. The resident should also be able to reduce stigma associated with the chemical dependence so as to comfort the family enough to establish a constructive working relationship.

COMMENTS : Family History

The family medicine resident should already be fairly skilled in conducting family interviews for the purpose of gathering needed historical information. Training them to gather substance abuse information is a good way to enhance their skills, since it is a sensitive topic for many families and taxes the doctor's skill more than gathering history about an appendicitis attack. The resident may encounter defensive reactions which may impede honest responses. Or the resident may encounter hostility. It is important that the resident learn to use skillfully the process of destigmatization in disarming the defensiveness so that the interview can proceed. It will also be important for the residents to determine how far they can reasonably go in various settings to obtain the needed information (i.e., how much information to get in one visit, whether to isolate the family members versus assemble the whole family for a certain line of questioning).

The confidentiality of substance abuse information is guarded by federal law which punishes breach of confidentiality by substance abuse programs quite severely [J*Anonymous, 1975]. At times, it is necessary to breach confidentiality, such as when reporting child abuse or neglect. In this case, there are guidelines for the best way to make the report without compromising the patient and one's own legal safety [Anonymous, 1979a]. In sharing information with the family, it is important to have at least verbal consent to permit family discussions of the illness and its manifestations. Without this, it may be impossible to affect therapeutic change or conduct conjoint clinical sessions. In collaborating with substance abuse treatment programs, consent forms will be necessary before they will talk with the physician, even to the extent of feigning ignorance of having any knowledge of a patient sent to them in referral.

The same schema for gathering historical information that is presented in Chapter 3 may be used with the whole family or any part of the family. Basically, one needs to gather information about intake, consequences, prior treatment and recovery attempts, and current motivation to recover. However, in assessing the family, one would not just focus attention on the person who ingests the chemicals. One would also assess the others in the family as to each of these major points.

STIGMA

Probably the first thing that the clinician must gauge is the degree of stigma that the family feels for having chemical dependency. Many times the stigma will interfere with psychological acceptance of the diagnosis and providing honest answers to the questions posed. One might ask whether they have ever known anyone with alcohol or drug problems, and then ask for their feelings about this person. This will provide clues to how they see themselves.

INTAKE

In chemically dependent families, often several members ingest chemicals [J*Abion, 1980; J*Alexander & Dibb, 1975; J*Attardo, 1965; J*Behar et al., 1983; E*Binion, 1979; J*Bohman, 1978; J*Cadoret & Cath, 1978; J*Cloninger et al., 1978; J*Cotton, 1979; J*El-Guebaly & Offord, 1977 & 1979; B*Goodwin, 1976; J*Goodwin, 1979; J*Goodwin et al., 1973, 1974 & 1977; B*Gurling, Clifford & Murray, 1981; J*Harbin & Maziar, 1975; J*Jacob, Dunn & Leonard, 1983; B*Kaij, 1960; J*Kaufman, 1980; B*McCrary, 1982; J*Rhydelius, 1981; J*Rosenberg, 1971; J*Schulz & Wilson, 1973; J*Schwartzman, 1975; J*Seldin, 1972; J*Stanton, 1978; J*Stanton et al., 1978; J*Steinglass, 1979 & 1981; J*Triplett, 1978; J*Whitfield, 1984; J*Woititz, 1978; J*Wolin et al., 1979 & 1980; J*Wolk & Diskind, 1961; B*Ziegler-Driscoll, 1979; J*Ziegler-Driscoll, 1977]. Quite often, one member of the family is singled out as the "worst" affected alcoholic or drug abuser, and the others are considered by the family to be "social" or non-problem users [J*Bowen, 1974]. The dilemma for the recovering chemically dependent person is that as he or she begins to learn about the illness, it is likely that he or she will begin to notice the same problems in other family members. Yet confrontation of these other family members may generate defensive denial and hostility towards the patient and his or her treatment. In some families, the identified substance abuser has had a role of being in the spotlight as the "family deviant" and the recovery of this person makes others in the family vulnerable to assuming the "family deviant" role now that it is vacant. In other families, there is so much substance abuse among its members that a recovering abstinent substance abuser is suspect of being deviant, and family members try to encourage relapse.

Often the chemical use by other family members differs qualitatively or quantitatively. For example, the daughter of an alcoholic man may smoke marijuana excessively, or his wife may drink moderately on top of several simultaneous sedative prescriptions she has obtained from various specialist physicians. His daughter may deny that pot is harmful, and his wife may claim that her drinking is moderate and that her drug use is medically approved and necessary.

In addition to obtaining drug ingestion information about the entire family, it is important to simultaneously obtain information about the attitudes of family members towards drug use and abstinence. Such an inquiry may also unearth other recovering chemically dependent family members or others who abstain or use chemicals with caution. Such family members may be more supportive of recovery than heavy users.

CONSEQUENCES

One may obtain far more extensive information about consequences of drinking or other drug use from family members than one might obtain from the chemically dependent person. Usually it takes one to three years of active recovery before the recovering

chemically dependent person can adequately assess the extent of damage the drugs were causing. But often it is possible to get more information about health problems, job difficulties, relationship conflicts in the family and social network by interviewing other family members. If the interview is done without the chemically dependent person present, the spouse or children may be more honest and forthright than if the drinker or other drug user is present and making faces or arguing about every point. On the other hand, a conjoint interview at some time may be very helpful in convincing the substance abuser that there is a heap of trouble that is associated with the addiction (see Chapter 4).

One also should obtain information about the consequences that have arisen in the family as part of the adjustment to the addictive disease, such as the roles that various family members have accepted. For instance, a child may have missed a lot of school because he was worried that his mother would fall and hit her head on days when she was quite intoxicated. Or a wife may have developed sedative tolerance by drinking along with her husband so as to protect him from being harmed at the bars where he drinks or on the way home late at night. This same wife may have neglected the children in order to enact her role as CHIEF ENABLER [A*Soft; B*Liepman, Wolper & Vasquez, 1982]. The residents may utilize the Family AFGAR Index [J*Good et al., 1979; J*Smilkstein, 1978] to screen for family problems.

Consequences of drinking or taking drugs are not all negative. While there undoubtedly will be many harmful sequelae from the addictive disease, there are usually several important positive consequences of the chemical ingestion which will disappear during early recovery, much to the chagrin of the family. These will constitute side-effects of recovery. They may be so difficult for the family to weather that relapse or family crisis may develop. In some cases the family will break apart during recovery; in other cases, another family member will become symptomatic and will replace the missing functions. A case example may help to illustrate this:

John and Mary Jones have been married for 22 years and they have three children: (a) George, a college senior in another state, (b) Tina, a freshman at the local community college who lives at home, and (c) Carolyn, a highschool junior. Mary has been recently detoxified from her excessive use of alcohol and tranquilizers that she was getting from her gynecologist, psychiatrist, and neurologist. After going through a rehabilitation program, she has now joined AA and has abstained from chemicals for three months. She claims to be doing quite well, but she complains that her family is having a hard time and she is worried about them. When she was abusing the tranquilizers, Tina had been running the house, shopping for groceries, caring for Carolyn and John. Despite all the added responsibility, she had done well at school too. Now, Tina seems to have problems with all the time she has on her

hands. She was socializing for the first time in her life, but it seems that she is choosing the wrong kind of friends. She gets drunk quite often, not returning home until very late at night, and her grades are slipping at school to the point that she might flunk out. John seems to be no help with this problem. He has absorbed himself deeply in his work and just shrugs his shoulders any time he is asked to discuss Tina's problems. (See also [H*Brooks, 1981].)

On careful inquiry, one discovers that during Mary's most heavy ingestion, Tina played the role of FAMILY HERO and obtained ego gratification from this. John did not participate significantly in the marriage partnership, as he found it far less stressful to deny what was going on at home and to bury himself in his job. Now that Mary was sober, she had reclaimed her responsibilities at home, denying Tina a gratifying role. In fact, Mary had resumed "mothering" Tina, which felt highly offensive to Tina after all these years of being a "parental child". During the early months of recovery, John and Tina found themselves angry at Mary, but afraid to express this to her because of their desire to support her recovery. But their behavior reflected their discomfort with the new arrangement. Without timely intervention, it is likely that John and Mary would eventually separate and Tina would become chemically dependent.

PRIOR TREATMENT

An assessment of prior attempts to obtain help or recover will provide insight into motivation, beliefs, skills and barriers which are likely to affect the current attempt at recovery. Families which have been (a) educated about chemical dependence, (b) destigmatized, (c) introduced to the self help programs (see Chapter 6) will be much further along than families which have experienced none of these. It is important to know exactly what treatment each member of the family had, including how much treatment and when. For instance, if John and Mary had been in marital therapy during her rehabilitation program, they might be communicating better than they are. But John was "too busy" to attend the marital therapy after the first session and never showed up. Mary had tried to quit the tranquilizers once before, at the urging of her psychiatrist. He was not aware that she was taking other tranquilizers and alcohol in addition to the benzodiazepine he was giving her. At first she did very well, by increasing her doses of other drugs and alcohol. But as she ran low on supplies of the other tranquilizers, she began to drink more. Noting that she was becoming like an alcoholic, she quit drinking altogether, developed the shakes and insomnia, and returned to her psychiatrist like a "basket case". He promptly restarted her tranquilizer and never again suggested that she quit.

The family physician also needs to obtain information about other help sought, even if it was not strictly for chemical dependence. John and Mary had seen a psychiatrist once for sex

problems. After the first two sessions, John stopped attending, but Mary continued for three years. She convinced the psychiatrist to give her a minor tranquilizer for dealing with the frustration of her husband and panic attacks which she described. Carolyn had been referred to a school counselor for clowning around in class. She did not spill the beans about her home life problems until the third referral in two years, and then the counselor felt unable to get involved. The referrals stopped. George had also been in trouble in high school for possession of drugs and absenteeism. George was trying to earn some spending money by selling some of his mother's tranquilizers. He landed in the juvenile court, but John got a lawyer who was able to arrange a deal that let George off without even probation. Each of these episodes of trouble were connected with the chemical dependence in the family. Yet no one recognized them as such, so the help missed the mark. By the looks of the history of treatment shown above, the Jones family seems to always come out on top, manipulating the therapy or community agency to avoid change. It is likely that the same will happen in the future unless the therapist can devise a way to overcome their fear of change.

CURRENT MOTIVATION

If the family physician knows how motivated the family members are to recover, he or she can plan the treatment more rationally. If there is a lot of ambivalence in some family members, the treatment plan will need to depend more on others who are more solidly motivated to recover. Sometimes there is strong motivation among some family members towards a goal which may not be recovery [J*Ablon, 1980; J*Blechman, 1982; J*Jacob et al., 1981; J*Janzen, 1977; J*Kaufman, 1980; J*Koppel, Stimmler & Perone, 1980; J*Little & Pearson, 1966; J*Mead & Campbell, 1972; J*Meeks & Kelly, 1970; J*Miller, 1980; J*Noone & Reddig, 1976; J*Rosenberg, 1971 & 1981; J*Smilkstein, 1978 & 1980; J*Stanton, 1979; J*Stanton et al., 1978; J*Steinglass, 1979 & 1981; J*Whitfield, 1984]. If there is motivation to avoid negative consequences, then the treatment plan may need to include some coercive contingencies. For example, John wants his wife to recover, but he sees no need to change his behavior. He might have come to the marital therapy that was offered if the program had threatened to discharge Mary prematurely due to his absence. Had Carolyn's school counselor or George's case worker at the juvenile court insisted upon a family evaluation as a condition of avoiding school suspension or incarceration, respectively, it is likely that they would have had better compliance (see Chapter 4). If there is motivation to achieve some ancillary goal such as better sexual relations, improvement of the child's school performance, or less arguments in the home, then the treatment plan may be devised to demonstrate the role of the chemical dependency as a factor hindering achievement of the family's stated goal.

TEACHING HINTS: Family History

Have the residents read about taking history from chemically dependent families [B*Liepman, 1983 & 1984]. Have the residents view a videotape [A*Family Secrets; A*Identifying the Alcoholic Patient] or watch an instructor interviewing a chemically dependent family or some of its members. Have the residents critique the performance, using what they have been taught about what historical data one needs to gather.

Have the students interview a family, or part of one, in a group setting using a volunteer recovering family. Have the volunteer family select a time prior to recovery so they can pretend that it is then when answering the questions. Have one student at a time do the interview while another serves as a coach, whispering suggestions in the ear of the interviewer. Have a third student observe and record content gathered while a fourth observes and records the interpersonal process (i.e., non-verbal communications). After a specified time interval (5 or 10 minutes is usually enough), stop the interview for feedback from the observers. Then rotate tasks and resume the interview for another time interval, continuing this process until everyone has had an opportunity to play an active role at each task. If the group of students approaches ten, it might be better to split into two groups with two families, as four or five trainees per group is ideal.

Using the role play technique, have the students try to take an "awful" history, purposefully constructed to demonstrate the wrong way to go about gathering this sensitive information. Because everyone knows this is just for fun, they can act quite unprofessional. But it will sensitize them to "wrong" approaches when they see it in themselves or in their peers and clinical role models. Be certain to include the feedback exercise interspersed frequently between interview sequences. The recovering chemically dependent family who volunteered should know that the students are trying to do the history wrong, and should participate in the feedback so that the students hear what impact their "wrong" approach had on the family's feelings and the doctor-patient relationship. If there is not a sophisticated recovering family available to participate in this exercise, the students themselves and faculty might play family roles. At least 10 students would be necessary to do this.

Have the residents use the Family APGAR Index [J*Good et al., 1979; J*Smilkstein, 1978] to screen for family problems in twenty consecutive families in their practices. Then have the residents interview the families that got a low score to determine the nature of their problems. Or have the residents ask a few of the troubled patients in their practice to record daily health diaries [R*Freer]. Then have the residents review the diary contents to see what role alcohol and drugs play in their patients' lives.

Have the students debate about whether to gather a substance abuse history or parts of it from the whole family versus separately from various family members in different clinical situations. Several cases might be used as examples.

1. an adolescent with marijuana and alcohol problems,
2. a single mother of two who drinks and takes multiple tranquilizers,
3. a father who drinks too much and a mother who drinks with him,
4. a grandmother who drinks in combination with her multiple medications,
5. a hermit who drinks too much according to his niece,
6. a pregnant opioid addict who is living with her dealer-boyfriend.

EVALUATION: Family History

Observe the resident taking a history from members of a chemically dependent family, whether during a clinical encounter (on video or live observation) or fabricated for evaluation purposes using a volunteer recovering family pretending to be pre-recovery. Determine how thorough was the history and observe the interpersonal process to identify any nonverbal communications emanating from the resident that might interfere with gathering a complete substance abuse history from the family.

FAMILY EDUCATION

LEARNING OBJECTIVES: Family Education

The resident will demonstrate the skill of educating patients and their families, clearly and sensitively, about the addictive diseases. The resident will be aware of deficiencies in educational programs to which patients and families have been referred so that the knowledge may be supplemented by the resident or other members of the health care team.

COMMENTS: Family Education

The lay public believes lots of myths about alcoholics, alcoholism and alcohol, drugs and drug abusers, tobacco products, smokers and addiction to smoking. The physician is a respected medical authority that patients expect to have accurate information about health problems. As long as the patient believes that the addictions are medical problems, patients are willing to believe almost anything the physician says as long as it fits with their experiences, makes sense and is stated with confidence. If a physician relies upon lay mythology rather than scientifically based medical information, the patient will not believe that there is anything new to learn about the problem.

THE DISEASE MODEL

The physician can begin to demythologize the addictive disorders by explaining about the disease model of chemical dependency. Historically, the addictions have been classed as voluntary deviant and immoral behaviors [E*Brecher, 1972; B*Ray, 1978]. Our laws and our language reinforce this model (e.g., calling a person who has a drinking problem "a drunk" comes from "drunkard" both of which dehumanize the person suffering from the disease alcoholism; and calling the person who is recovering through abstinence a reformed alcoholic suggests connotations of criminality and sinfulness). In teaching the family about the disease model, it is often necessary to explain the history of their beliefs to help them realize that their ancestors were ignorant because that was all that was known at that time and that the new disease model is a result of medical progress in recent years. It usually helps to share with them some of the new information we have about inheritance of metabolic factors such as the sluggish acetaldehyde catabolism [J*Schuckit, 1979], the THF story [B*Blum, 1982] connecting alcoholism with opiate receptors, the endorphins, the benzodiazepine receptors, nicotine metabolism, etc. so that they begin to understand that there is much more to addiction than ingesting a drug.

It is often helpful to share with patients and their families the consequences they have suffered which can be attributed to drug induced damage or impairment of function and health. It is often surprising to victims of chemical dependency and their families that many of their life troubles are related to the chemical ingestion and the surrounding addictive behaviors [B*Brooks, 1981]. It is also helpful to outline the risks of future consequences that may be suffered if recovery is not prompt and successful [B*Meyers, 1983; B*Ostrea, Chavez & Stryker, 1978; B*Fattison & Kaufman, 1982; B*Robe, 1982; B*Wartenberg & Liepman, 1984].

It is important to explain the chronic nature of the addictive diseases so that victims and their families understand that there is no simple "cure", rather that remission can be achieved only by continuing devotion to a life-long recovery program. They need to understand the danger of relapse in facilitating further progression of the disease and its consequences. They also need to understand the importance of prompt intervention when relapses occur.

Explaining how the family participates in the disease seems to facilitate engaging family members in treatment and recovery. Often the easiest concept to communicate to the family is their victimization by the disease. This is, however, often the hardest concept for the drug abuser to accept. One must also convince the family and the drug abuser that recovery requires changes which the whole family must share. The issue of responsibility for one's actions is often a major point of confusion and controversy in chemically dependent families. Since there is usually a lot of blaming and guilt floating around the family, it is important to put things in proper perspective. One can teach that the person

who ingests the chemicals has a disease which he or she did not intend to catch (this reduces stigma and guilt), that the person is permanently disabled from being able to safely use the chemical and its sister chemicals again in the future (this sets the stage for an abstinence goal of recovery), that the disease needs long-term treatment (this offers hope and prevents simplistic notions of magic cures), and that the family also suffers from a related behavioral disorder (i.e., compulsive enabling [A*If You Loved Me, A*I'll Quit Tomorrow, A*The Enabler, J*Whitfield, 1984]). It is usually a relief (of guilt) for family members to hear that no one is solely responsible for the development of the disease, but it is harder to convince each individual to take responsibility for his or her part of recovery. How this is said may be critical in obtaining family participation in recovery. Total guilt reduction may lead to total apathy towards treatment, on the other hand, guilt induction may promote continued denial and active avoidance of treatment. Only the middle position (i.e., you did not cause this disease, but you can help in the recovery, you did not intend anyone harm by your actions as an enabler, but you need to stop the enabling behavior in order for the whole family to get well) seems to promote participation in family-oriented treatment [B*Nirenberg & Liedman, 1984].

GOALS OF RECOVERY

It is also important to explain goals of treatment and recovery to the family. Usually there are several important issues that need resolution in CD families.

One goal of recovery is developing a different pattern of use of psychoactive abusable drugs. Most chemically dependent patients desire a return to non-problem drinking or drug use as a goal of treatment. In the United States, more so than in Europe, there is strongly held dogma among most recovering alcoholics and alcoholism treatment professionals that recovery is impossible without total abstinence [J*Goodwin et al., 1979; J*Pendery, 1982; J*Tournier, 1979; B*Zimberg, 1982] from alcohol and other mood-altering abusable drugs. Many in the drug abuse field believe this too, though advocates of methadone maintenance and other long-term chemo-substitution approaches disagree [B*Bolter, 1979; B*Brecher, 1972; B*Cushman, 1981; B*Freedman, 1979; B*Hofmann & Hofmann, 1975; B*Lasagna, 1979; B*Ray, 1978; B*Soldatos, Kales & Cadieux, 1979; B*Stunkard, 1979, B*Ungerleider, 1979]. Families who believe that cutting down intake is possible and an acceptable goal may feed into the chemically dependant person's fantasy of becoming a controlled non-problem user once again. This could undoubtedly create a philosophical conflict in the relationship between therapist, patient and family unless all parties agree philosophically. Without treatment, it is unusual to successfully reduce drinking or other drug use to a safe level for an indefinite period of time, though most treatment professionals know one or two people who appear to have recovered in this way [J*Davies, 1962]. With abstinence oriented therapist, the usual outcome of this philosophical conflict is patient and family alienation from treatment and community supports (i.e.,

AA, Alanon). Many patients maintain denial of having alcoholism in this way, opting to use formerly unreliable "will power" to control drinking or other drug use. Many suffer episodic relapses, which result in family anger and resentment, personal intrapsychic depression or demoralization. If this causes the patient and family to abandon their previous insistence upon recovery by controlled use, this is called humility or hitting "rock bottom" in AA. However, if it prompts the patient or family to abandon recovery, it is a shame. In most cases there is history of numerous prior instances of unsuccessful attempts at intake reduction and control which demonstrate the futility of trying to return to controlled non-problem use. If the patient returns to an abstinence oriented therapist after relapsing while trying to control drinking or other drug use, most therapists will welcome the patient back and use this failure as an opportunity to reiterate the importance of abstinence in recovery.

A minority of North American alcoholism treatment professionals, in concert with many European experts, advocate teaching selected patients to control their drinking [J*Sobell & Sobell, 1973 & 1976]. There has been recent public airing of scientific controversy over the question of whether such treatment constitutes an unethical practice because it delays "real treatment" and hence indirectly may cause the patient and family to suffer additional harm during the delay [J*Marlatt, 1983; J*Peele, 1983]. Harsh scientific criticism has been leveled at the Sobells for relying upon self-reports of alcoholic subjects in their earliest study, upon which subsequent study was based. Evidence has been gathered showing that very few of the patients who were exposed to the controlled drinking subgroup of their earliest treatment outcome study have recovered over the decade since the treatment [J*Penderg, 1982]. However, the comparison group exposed to traditional abstinence oriented treatment was not exposed to the same scrutiny by Penderg. According to recent more sophisticated studies, the Sobells report that patients exposed to controlled drinking training exhibit significantly fewer drinking days and better global adjustment than the traditionally abstinence trained patients [B*Heather & Robertson, 1981; D*Marlatt & Gordon, 1980 & 1984; B*Sobell & Sobell, 1978]. Since traditional treatment approaches to alcoholism treatment are far from universally effective, criticism of novel approaches in the laboratory seems unfounded, as long as proper informed consent procedures are used. However, until the controlled drinking (or other drug use) treatment approach has shown unequivocal long-term effectiveness with all, or a definable subgroup of, chemically dependent patients, it should be considered to be an experimental treatment requiring more scientific study. At this time, it seems most prudent to recommend abstinence oriented treatment to all patients with prior history of failure to achieve non-problem drinking or other drug use, unless (1) the patient adamantly refuses to consider abstinence, (2) there are no serious contraindications to alcohol or other drug use other than those caused by loss of control over intake (e.g., serious cirrhosis of the liver, epilepsy, anticoagulation), and (3) there is a therapist available locally who has been thoroughly trained in the controlled drinking approach.

Successful recovery through controlled non-problem drinking may be achieved through development of substantial "control rituals" [J*Zinberg & Jacobson, 1976]. As long as the control rituals are applied, they may delay relapse. However, if drinking or other drug use continues after control rituals are relaxed, relapse becomes more likely. One advantage of the controlled drinking approach to those trained in it is the ability to participate socially in drinking situations without feeling awkward. A disadvantage is the alienation that most people will encounter from AA members. On the other hand, it may be easier for most persons suffering from alcoholism to permanently and successfully abstain than to control their drinking, especially if they are doing so without training or if their treatment taught them to abstain. Such individuals have the option of participation in an alternate society (AA) and ease of recognizing relapse in exchange for self-exclusion from drinking activities in mainstream society. Family beliefs about recovery may facilitate or hinder it, depending on whether the patient and family philosophies agree or disagree concerning abstinence.

Recent clinical outcome studies in the alcoholism field have suggested that outcome of recovery must be measured using many different indices of recovery [B*Maisto & McCollam, 1979]. When this is done, mixed results are often found [B*Moos, Cronkite, & Finney, 1982]. Moos and Billings [J*1982] showed that children of recovering alcoholics show marked improvement in depressive symptoms compared to their peers whose parents still were drinking alcoholically. The children from recovering families appear to be better off than children whose parents have never had alcoholism. One also finds improvement of psychological function (MMPI) of recovering alcoholics after four years of abstinence [J*Pettinati, Sugarman & Maurer, 1982]. However, another study found significant social and psychological distress in recovering alcoholics [J*Finney, Moos, & Mewborn, 1980]. Still another found general improvement in overall health in patients with reduced drinking patterns without total abstinence [J*Polich, Armor, & Braiker, 1980]. Clinically, if a person who suffers from chemical dependence quits using the drug, that alone may not suffice to produce dramatic improvement in all spheres of life. It may be necessary to provide a host of other supportive and rehabilitative measures to ensure full and satisfying recovery and to maximally guard against relapses. For example, an alcoholic who quit drinking because of a peptic ulcer may find that working in a brewery no longer suits him, so he begins taking disulfiram, attending AA and quits his job. While his duodenum may appreciate his abstinence, the increased nicotine intake at smoke-filled AA meetings and worries about financial problems may exacerbate his ulcer. Only a wholistic approach is likely to be successful in total recovery from chemical dependence.

Another goal of recovery is healing the family hurt that has arisen from the deceit, topsy-turvy priorities, broken promises, bringing shame upon the family, and the fear of death of the drug abuser. The family must make amends for these among themselves. To do so, they must realize that these problems are a part of the addictive process, lest they blame it all on the addicted person.

This is often done best by multiple family groups or groups for the family members of addicted persons (e.g., Alanon, Alateen) [J*Cadogan, 1973; J*Hendricks, 1971; J*Hirsch, 1961; J*Howe, 1974; J*Jansen, 1977; J*Kaufman & Kaufmann, 1977; J*Olsen, Russell & Sprenkel, 1980; B*Pattison & Kaufman, 1981; J*Rosenberg, 1981; J*Stanton, 1979; B*Steinglass, 1977]. These groups educate and reduce stigma simultaneously. As members self-disclose their own situations, others hear familiar stories and begin to understand that their own situation is not altogether unique. The sharing of stories becomes a means of affectively bonding together as a mutual support network and also permits members to reframe their experiences with the chemical dependency as elements of an illness. This process may also help family members learn to recognize and stop their own "enabling" behaviors, and therefore helps them to better support the recovering person's abstinence.

PROCESS OF RECOVERY

The family must learn about the treatment process. They must realize that recovery takes a long time, is an on-going life-long enterprise, involves the whole family, and has other benefits other than merely the cessation of drug ingestion. Myths that are common among the lay public suggest that recovery is merely will-power to stop, that the addicted person acts alone, that once he or she stops, the addiction is gone, and when relapse occurs, that the addiction starts over again from the beginning. In some cultural groups, recovery is deemed impossible (e.g., Puerto Ricans believe that if someone uses heroin even once, that the person is permanently hooked on it) [J*Ablon, 1980; J*Ablon & Cunningham, 1981; J*Benjamin & Benjamin, 1981; J*Gordon, 1981; J*Vaillant, 1966; J*Waddell, 1981; J*Walker, 1981; J*Westermeyer, 1981]. Some cultures view the abstaining person as weak or effeminate. Northern European cultures teach males to refrain from expressing emotions except when drinking, so abstinence in a recovering male of Northern European extraction would interfere with healthy emotional ventilation. In recovery, families must alter their interpersonal behavior, their personal management of feelings, and their attitudes towards alcohol, other drugs, chemically dependent persons, abstainers, honesty and dishonesty. They usually reap rewards from these changes as they regain personal resources, grow emotionally and spiritually, and improve their social milieu. But there may be an initial period of recovery when it feels worse to be abstinent than it does to be intoxicated, when life seems rougher than one can bear.

TEACHING HINTS: Family education

Have the students observe a lecture series at one of the local substance abuse treatment programs that is offered for patients and their families. Afterwards, have them discuss the salient topics and affective elements of the educational program that would be important to achieve in other similar programs.

Have the residents educate several families in their practice who have a substance abuse problem. If possible, have the several families attend a group educational session together, for cost-efficiency and taking advantage of the sharing of experiences which is so important. If more than one resident wants to merge patients and families from their practices, they might benefit from joining forces to provide the educational series. If expertise is lacking, they might solicit help from a substance abuse expert at one of the local treatment programs in putting on the series. Faculty participation or observation might enhance the quality of such a program by offering formative evaluation feedback while there is still time to try alterations in content, format, and style.

EVALUATION: Family education

Observe the quality of the education program offered to the resident's patients and families. Quiz the student on the high-points of a patient education program on alcoholism, smoking, or other addictions.

FAMILY COUNSELING

LEARNING OBJECTIVE: Family Counseling

The resident should be able to respond to overt or covert requests from the family for help with a chemical dependency problem. If the family is in treatment but fails to make progress in a timely fashion using the resources they have, or if the chemically dependent person relapses, the resident should be able to help the family to deal with this situation constructively by improving family utilization of resources or by supplementing them. The resident should be able to ferret out and recognize negative side-effects of treatment and to respond to them constructively to prevent relapses, treatment failures, or irreversible untoward consequences of treatment. The resident should develop specific skills needed to work effectively with chemically dependent families, including:

1. Assessing the recovery program,
2. Reducing stigma,
3. Enhancing motivation,
4. Prescribing changes in recovery program,
5. Monitoring progress.

COMMENTS: Family Counseling

It is beyond the scope of this monograph to provide guidance on family counseling in detail [B*Berenson, 1979; B*Doherty & Baird, 1983; B*Dulfano, 1978; B*Howard & Howard, 1978; B*Kaufman & Kaufmann, 1979; B*Liepman, 1984; B*Nirenberg & Liepman, 1984;

B*Paolino & McGrady, 1977; B*Pattison & Kaufman, 1981; B*Stanton et al., 1982; J*Steinglass, 1979; J*Steinglass, Davis & Berenson, 1977; B*Liepman, Williams & Whitfield, in press; B*Zimberg, 1982]. However, this section will focus on specific skills that are necessary for successful family counseling of chemically dependent families.

ASSESSING A RECOVERY PROGRAM

One can gather historical information about the efforts the family has tried in order to recover from chemical dependence. In analysing what is found, one may discover that many of the efforts were futile because:

1. the family chose poor quality recovery resources,
2. did not allocate sufficient effort to their choices,
3. neglected certain of their important needs in treatment planning, or
4. temporarily succeeded with the help of their choices, but prematurely abandoned them.

This helps the family physician to discover why the family has not recovered previously. It may predict future patterns of family resistance to utilizing resources that are made available and alert the family physician to the need for family education or motivational counseling. Prior treatment history may also suggest the existence of special needs of the family that were not previously noted or considered important. For example, John and Mary Jones sought help from a psychiatrist friend of theirs who drank (alcoholically) with them. He was unable to help them with their chemical dependency problems because his own personal life was out of control too, and his personal denial had extended into his professional life (see Chapter 9). Their choice of a professional was inadequate for their needs. And they did not follow through. After only two sessions, John quit coming (perhaps appropriately). But when Mary finally got treatment for herself at the inpatient rehabilitation program, she did not get help for her family. John only attended the first marital session in Mary's rehab program. The children did not participate in family education, sharing of feelings with other children, and planning of behavioral change for Mary or the rest of the family. When Mary joined AA, her family did not join Alanon or Alateen. Hence, in summary, the Jones family missed out on lots of the resources that were available to them and failed to follow through on some of the efforts that they started.

REDUCING STIGMA

The primary goals of stigma reduction are to discourage placing blame and distract attention from past behavior in order to encourage change and involvement in current treatment [J*Rowenberg, 1981]. Since stigma generally elicits "defensive reactions, reduction of stigma should permit entry of new ideas and experimentation with new behaviors.

Stigma comes both from within the person and from the environment. The personal origin of stigma is attitudes towards chemical dependence which were taught by parents, teachers, public authorities and peers in the past. These attitudes became internalized as beliefs which are often hard to counteract. By teaching an individual that these attitudes reflect an archaic set of beliefs which have since been discarded in light of modern scientific knowledge, most persons will begin to reexamine these attitudes. Some persons will have beliefs based on religious training that drinking alcohol is sinful (e.g., Jehova's Witness, Mormon, southern Baptist, Moslem). These persons may disagree with the philosophy of the physician who makes a distinction between drinking and alcoholism. But one can approach them with the notion that the addictive disease makes it hard for the patient to avoid sinful behavior.

Stigma from environmental sources, from family, friends and even professionals, may reflect ignorance. The CD family can be inoculated against these harmful influences by education and by talking with other peers who have undergone recovery. The positive alternative environment of the self-help communities or others in a treatment program tends to help.

ENHANCING MOTIVATION

Motivation to recover is often the major missing ingredient in chemical dependence cases. Lack of cooperation or participation by the patient or family can make even the best treatment unsuccessful. Defensive denial may play an important role in lack of motivation. The family may outwardly deny that: (a) there is anyone in the family who has problems of any sort, (b) the family's problems are secondary to chemical ingestion, (c) the pattern of drug use in this family is problematic, (d) the problems are chronic rather than transient, (e) treatment is necessary for recovery, (f) recovery is possible, (g) treatment is worth the investment, or (h) certain components of treatment such as abstinence, family therapy, aftercare, or involvement in a self-help program may facilitate recovery. When ignorance produces these beliefs, the physician may successfully counteract them by education. But when these beliefs are refractory to educational efforts, they probably reflect fear of change in the status quo. In this case, the family physician may need to address the advantages and disadvantages of change.

Sometimes the patient or family will respond to concerns about a serious medical, psychiatric or social danger. If they present in crisis, they may be channeled into treatment while the crisis is still a motivating force [B*Liepman, 1984]. Families tend to be more receptive to advice during a crisis than during any other time [J*Smilkstein, 1980], probably because they are made uncomfortable during a crisis, and they begin to believe, although transiently, that their usual responses are inadequate. What scares one person may not scare another. One patient may finally be motivated by an experience of near death, another by acute embarrassment at work; one may be motivated by a threat of

divorce, another by the alcoholic amnesia, or "blackout," and still another by an eruption of violence. It is very important for the family physician to learn to recognize a family crisis, and to use it aggressively as an opportunity to affect rapid change before the family "settles down" again to their usual *modus operandi*.

In order to motivate chemically dependent families, one may need to have resources ready to use at a moment's notice for diagnosis, education, detoxification, rehabilitation counseling, and self-help. A delay in offering these to a family may be all it takes to lose the opportunity. One study has shown that wives of alcoholics wait an average of 7 years after the first obvious occurrence of problem drinking [J*Gallant, 1981, J*Corman & Rooney, 1979] before seeking help. In this study, pressure from the family physician led the wife to seek help within 2.7 years, on the average! If Smilkstein is right, the family probably waited until another crisis to seek help.

Motivations for quitting are in direct conflict with motivations for not quitting (or for relapsing). A person who relapses may weigh the consequences of drinking and not drinking, and decide to risk the consequences. Some experts feel that this occurs only with the help of a large dose of denial, but others recognize the very real benefits that drinkers, drug users, and smokers perceive in association with the ingestion of their favorite chemicals. Failing to quit when dangerous consequences arise may be due to fear of the results of quitting, such as physiological, psychological, or social withdrawal. Quitting may not be easy or even tolerable for a person who attributes such an advantage to relapse unless treatment provides new alternatives never before encountered.

Concerns and complaints expressed by people in the patient's social environment (e.g., family, employer, friends) are often insufficient to motivate a person to seek treatment. This may be a result of counterbalancing acceptance and support of the drinking or drug use by others in the social environment [J*Koppel, Stimmler & Perone, 1980; B*Liepmann, Wolper & Vasquez, 1982]. Even the complainers are usually ambivalent, because the drinking or drug use serves some useful purpose within the interpersonal process (e.g., the spouse can feel superior and make family decisions without negotiation or compromise; the alcoholic can feel independent and powerful; the oldest child can have an opportunity to act as an adult in the family). These "advantages" are often denied by the people involved until late in the treatment. Complainers usually focus on the "disadvantages" of the chemical abuse. However, if there were only disadvantages without counterbalancing advantages, the substance abuser and the enablers would be much more likely to stop than they are.

A spontaneous crisis will sometimes motivate an addicted person to recover. But spontaneous crises can get risky. People die, get divorced, become permanently disabled, are institutionalized, and so forth, during spontaneous crises. Although it is to the advantage of the doctor to wait until nature takes its

course, and the next crisis occurs to motivate the patient and family to request help, this is not very safe for the patient, family, or the public in general. It is also possible that the next crisis will occur at a bad time (e.g., when the doctor is on vacation; when the patient and family are out of town; when all the detox program beds are full), or the crisis may be the sort of thing that the family physician is not comfortable treating (e.g., a family member becomes suicidal, or the patient fractures his skull). Natural crises do not fit neatly into one's schedule.

Rather than wait for a natural crisis, the family physician can have greater control if he or she designs and engineers an artificial crisis by bringing to a boil the already brewing discontent and concern of the people in the patient's social environment. The spontaneous crises occur when social, psychological, and physiological processes go beyond the homeostatic capacity of the person and family to withstand them. During the interim periods between crises, there is nevertheless subclinical social, psychological, and physiological malaise that can be highlighted to motivate early acceptance of treatment. The procedure is called "intervention" [Johnson, 1973] or family constructive coercive confrontation (FCCC) [J*Gallant, 1981; B*Liepman, Wolper & Vazquez, 1982]. It involves gathering together the influential people from the social network of the chemically dependent person to form a team with the goal of confronting the illness and encouraging acceptance of treatment. (See Chapter 4 for more detail.)

MONITORING PROGRESS

The recovery process is long and complex. As the family adjusts to life without chemical assistance, the physician may notice constructive changes in interpersonal and intrapsychic function of family members. However, it is just as likely that the family physician will notice family distress in the form of emotional or psychosomatic problems. The problems may occur in any family member, not just the recovering substance abuser. By keeping close collaborative contact with the substance abuse treatment team, the family physician may be able to discover the origin or etiology of the presenting problem in the current stage of the therapeutic process. For example, Lisa's sudden onset of dysmenorrhea coincides in time with the return of her mother from an alcoholism rehabilitation program. As her mother resumed maternal household responsibilities, she displaced Lisa from a comfortable niche. Now free many more hours than ever before, but unable to suddenly form a new identity as an adolescent girl, Lisa found solace in the sick role which provided (1) relief of competition with her mother for household chores, (2) relief or pressure to become active in school activities, and (3) an opportunity to obtain some long-overdue nurturance from her mother. The family physician can utilize this information to more sensitively deal with the problems presented by the family.

If it appears that the family is about to terminate treatment prematurely, the family physician may attempt to do some trouble-shooting to determine the reasons for this. At times, inadequacy of the treatment plan or team may cause the family to leave treatment. In other cases, the family is resistive of the pressure to change imposed on them by the treatment team. In such cases, the family physician might help by showing the family how well they are progressing and encouraging them to stick with recovery.

ALTERING THE RECOVERY PROGRAM

If deficiencies in the original treatment plan or team are discovered, the family physician may prescribe changes. The changes may be suggested to the treatment team directly via collaborative links, or indirectly through the patient or family. For example, John was started on disulfiram after three brief "slips" during a stressful period of his recovery. This was discussed with the treatment team before the prescription was written, to be certain that it would not interfere with the treatment plan. The information about the relapses had not been offered to the therapist by the patient or family, hence the therapist began to slow down the pace of treatment and paid more attention to transference issues.

TEACHING HINTS: Family counseling

Ask each student to attend at least one closed Alanon, A.C.A. (Adult Children of Alcoholics), or AA meeting. After the meeting, the student should mix with the participants to inquire informally what ultimately motivated them to join.¹ What other serious crises had occurred in their lives that did not result in immediate recovery? Why did they not stop drug use sooner?² Alternatively, the student may do this informal survey in an alcoholism or drug treatment program, or the instructor may invite a group of recovering patients or families to attend the class. Readings [B*Alibrandi, 1978; J*Glaser & Ogborne, 1982; J*Goodwin

¹ WARNING: Do not send students to do a formal survey at these confidential meetings. Students with pencil and paper taking notes would be understandably rejected by many regular participants in the self-help community, and might scare away newcomers. The proper approach is to have the students share, at the appropriate time, that they are attending as medical students or doctors who want to learn about AA/Alanon so that they can refer their patients to it. As they talk with individuals over coffee, before or after the meeting, they can ask questions as suggested above.

² Be certain that the students penetrate beyond such superficial, pat answers as, "stupidity," or "I was crazy," and so forth.

et al., 1979; J*Tournier, 1979; B*Zimberg, 1982] and film [A*Alcoholics Anonymous; A*If you loved me] about the self-help programs may spark lively discussion before and after visits by students to meetings. It may be useful to ask students to attend meetings on their own initially, then monitor their attendance. When few or none report having gone, one can indicate that their response to the referral was not atypical of the clinical response of most patients asked to attend self-help meetings. Then the students can be taken to some meetings by assigning them to "intake sponsors" prearranged through the instructor.

Arrange for the students talk with the families of some recovering substance abusers, so the students may learn of other reasons why these people may have become motivated. Let the students discuss among themselves the findings of their surveys.

If it is possible, have the resident videotape two clinical encounters with one family, both when the substance abuser is sober, and when chemical ingestion has recently occurred (i.e., the drug effect is still present). This might be accomplished by taping a family when a relapse has occurred and then taping another session when the substance abuser has completed detoxification. Study the two videotapes, contrasting the interpersonal behaviors of the family in these two states. If one can identify advantageous or desirable family behavior during the substance use tape, one may have identified one important reason for that family's resistance to recovery.

There are two good films that portray the use of social network coercion in motivating an alcoholic person to enter treatment [A*I'll Quit Tomorrow; A*The Intervention]. Have the students view one of these before embarking on discussion about the technique and ethical considerations. Have the students watch for enablers in the film to determine how they undermine the efforts of others who are trying to take a constructive approach. The students might prepare a group role-play exercise where a mock "intervention" is portrayed. Such an exercise takes approximately two hours with ample time for feedback and discussion breaks between segments. Expert coaching on this might be obtained by inviting a local alcohol or drug counselor who works with families.

If it is possible for each resident to follow at least one patient through a substance abuse rehabilitation program, the resident will have the opportunity to work collaboratively with rehabilitation counselors and other professionals. Otherwise, it might be helpful to have several staff counselors from a local agency hold an informal "rap session" about the problems and assets of teamwork between counselor and physician.

EVALUATION: Family Counseling

Direct observation in a clinical setting of resident interaction with the patient or family is the best method of evaluating counseling skills. One could evaluate both the content and the technique of delivery. Videotaped records of actual visits

permit the resident to observe his or her own behavior without the emotional pressure of the clinical situation. In addition, both the preceptor and the resident can study the interaction carefully to determine exactly what might be changed, what may have triggered undesirable responses, and what will need to be done on the next visit. The resident should be able to demonstrate stigma reduction, motivational enhancement, assessment and monitoring of a recovery program and collaborative working relations with other team professionals. The resident should demonstrate sensitivity to health problems in all family members as signs of therapeutic stress, both good and bad. The resident should remain attentive to signs of trouble in the recovery program of substance abusing patients and their families and know how to take corrective action.

Feedback from substance abuse professionals who are working collaboratively with residents or who had a student for a block rotation can be very helpful in assessing knowledge, attitudes and behavior.

CHAPTER 9: THE FAMILY PHYSICIAN AT RISK

By Joseph V. Fisher, Charles L. Whitfield, & Michael R. Liepman

OVERALL OBJECTIVES

When it becomes apparent that a professional colleague may have or be developing a problem which has adversely affected or could potentially impair his or her clinical function and/or personal life, the resident will offer to assist the colleague and his or her family constructively without undue delay. Such assistance will not facilitate progression of the problem and may utilize institutional programs to foster physician well-being. The resident will also periodically self-assess personal and family risk of developing substance abuse problems, and take specific measures to alter aspects of life style or chemical ingestion patterns that may be detrimental to personal well-being.

COMMENTS : Introduction

Since the early 1970s, there has been a growing concern about physician impairment. The incidence of suicide and psychiatric illness, chemical dependence (CD), and cardiovascular disease among physicians motivated some state and national organizations, (such as the American Medical Association, American Academy of Family Practice, the American Psychiatric Association, the [R*Center for the Well-Being of Health Professionals] and others) to direct their concern to them. There are now programs for the troubled, impaired, or disabled physician in nearly every state in the United States, which vary in size, structure, and quality [B*Anonymous, AMA, 1982; J*Clinger, 1977; J*Gitlow, 1977; J*Newsom, 1977; B*Robertson, 1981; J*Steindler, 1977; J*Talbott, 1977]. These programs primarily focus on motivation and rehabilitation of the impaired doctor, although their goal for the future is to improve early case identification and intervention before the problem reaches an advanced stage. Some organizations have begun to develop prevention programs to foster self-assessment and better risk management through education.

DEFINITIONS

The definition of physician impairment of the AMA housestaff committee [B*Tokars, Bremer & Peters, 1979] states that "a physician is impaired when personal problems interfere with patient care, education or personal life." Gitlow [B*Whitfield, Bissell & Wesson, in press] defines three classes of physician impairment:

1. the incompetent physician;
2. the malicious physician; and
3. the psychiatrically disturbed physician.

He also lists reasons for the impairment:

1. failure to recognize one's own physical disability;
2. psychotic or irrational behavior;
3. senility; and
4. addiction to drugs.

An important element in the chronicity of physician impairment is denial. When a physician is suffering a problem, the failure to recognize or accept the problem and its impact upon personal and professional life may interfere with problem solving and prevention of functional impairment. In essence, if a physician is suffering a chronic, recurring or progressive problem and does not seek or accept appropriate help, the problem is unlikely to go away and it may begin to impinge upon the professional sphere.

Many personal problems remain contained within the personal sphere, but some problems eventually spill into the professional domain. If a resident comes to work drunk or hung-over, it is quite obvious that personal life choices are affecting professional function. But some personal problems affect professional function only in a subtle manner because of the way in which the physician uses self in the human interaction with patients. For example, if the resident has struggled in vain for years to get his mother to stop taking tranquilizers, it is likely that the resident will experience familiar feelings while trying to convince an older female patient to give up tranquilizers. It would not be unusual for such a resident to become angry and frustrated at the patient's reluctance to abstain from these drugs; yet the patient might experience the doctor's reaction to her situation as overly hostile and unempathic.

Residency may be a time when transient physician impairment is so common as to be considered normative. Acute stresses caused by sleep deprivation, questions about identity as a physician, marital discord, financial problems, feelings of clinical inadequacy and depression have become synonymous with being a resident [B*Shem, 1978; J*Loes & Scheiber, 1981]. Issues such as problem patients, clinical errors, sexual overstimulation at work and deprivation at home, helplessness in overcoming death, ethical dilemmas, and clinical uncertainty may also significantly interfere with a sense of well-being at all stages of training, but especially during residency [J*McCue, 1982]. In addition, so much of the resident's experience is controlled by others (e.g., call schedules, rotation assignments, clinical care of complex or private patients) that the resident experiences a major loss of control over one's life [J*Frey, Demick & Eribace, 1981]. Some residents turn to mood altering drugs to help them relax, stay awake, or forget their emotional pain. Often this solution is promoted by their peers and teachers both informally and formally. The entire experience of residency training may be thought of as a transient episode of acute physician impairment early in professional life [J*Small, 1981]. The way that peers and superiors deal with this predicament of the resident affects its immediate outcome [J*Chappel, 1981] and may set the stage for responses to future episodes in oneself, the family or colleagues.

It is beyond the scope of this monograph to deal with all aspects of physician impairment or stress in detail. Hence, in this chapter, further discussion will be limited to substance abuse related problems in physicians.

EPIDEMIOLOGY

Is the prevalence of CD greater among physicians than others? The AMA estimates that 4-9% of all physicians are impaired by alcohol or drug addictions, or by mental illness at some time during life, and that 40% of these are impaired by alcohol alone or by alcohol and another drug [J*Anonymous, 1972b; B*Steindler, 1975]. Bissell estimates that of United States physicians, approximately 224,000 of the total 324,000 use alcohol, and that 13,600 to 22,600 are, or will become, alcoholic [J*Bissell & Jones, 1976]. This represents a 5-10% lifetime prevalence, which equals or slightly exceeds that of the general population.

The incidence of physician narcotic abuse is estimated at 1.5%; that of the general population is set at 0.003% [J*Anonymous, 1972b; J*Hall, Stickney, & Popkin, 1978]. This represents an alarming disparity! While in the past, most cases of opioid addiction could be attributed to iatrogenic addiction, more recent trends have included cases of recreational drug abuse which began before medical school [J*McAuliffe, 1984].

Several studies suggest that many impaired physicians are dependent on more than one chemical. Among 93 physicians treated at the Mayo Clinic, 47% were seen because of an active problem with alcohol, with other drugs, or both. Of 31 physicians treated at the Menninger Clinic's Alcohol Recovery Program, 35% were treated for alcoholism, 42% for abuse of alcohol and at least one other drug, and 23% had no alcohol dependence but were addicted to at least one other drug [J*Connelly, 1975]. Of about 50 chemically dependent physicians treated at the University of Maryland, over half had multiple chemical dependence, either concomitantly or sequentially [B*Whitfield, Bissell, & Wesson, in press]. In Quebec, of physicians "known" to be actively addicted to drugs between 1974-78, 13.5% abused opioids only, 19% abused only non-opioid drugs, 57% abused opioid and non-opioid drugs, and 11% drank with their opioids [J*Wallot & Lambert, 1984].

RISK FACTORS

Should not physicians know better how to avoid addictive disorders than laypersons? An exceptional knowledge of pharmacology does not confer immunity to the development of these devastating illnesses. Experiments in prevention of childhood drug abuse via drug education backfired producing more experimentation! Once addicted, no matter how skillfully physicians attempt to control or manipulate their chemical use, most find no lasting solution to their problems short of total recovery.

Are there identifiable predictors of physician impairment due to addictive disorders? Is it possible to change the course of events to prevent or decrease the morbidity by early recognition and intervention? Studies of chemically dependent physicians show the following to be some potential etiologic factors:

1. genetic contributions;
2. family history of chemical dependence;
3. childhood physical illness or emotional deprivation;
4. career choice;
5. maladaptive response to stress during education and active practice.

Extensive studies of genetic factors in the human transgenerational perpetuation of alcoholism [B*Goodwin, 1976] suggest that these factors, to which physicians could not be immune, might play a significant role in physician impairment. Family rituals have also been shown to play a role in the transgenerational transmission of alcoholism [J*Wolin, Bennett & Noonan, 1979], suggesting that physicians might also be influenced by family of origin drinking history and rituals.

One report [J*Stout-Wiegand & Trent, 1981] suggests that easy access to narcotics, often given as the reason for the high rate of narcotic abuse among physicians, may not be a valid cause of this problem. This investigation showed that among physicians, dentists, and veterinarians (all of whom had access to narcotic drugs), the incidence of narcotic abuse was much greater among physicians than in the other two groups. Extensive discussion of role strain of physicians can be found in Gerber [B*1983] and Shem [B*1978].

Several retrospective investigations of physician impairment and one prospective study provide information about the influence of the families of origin on future physicians. A long-term prospective study of Harvard undergraduates [J*Vaillant, Sobowale, & McArthur, 1972] discounted role strain when they wrote "that only those physicians with the least stable childhood and adolescent adjustments appeared vulnerable to these occupational hazards." A retrospective study of addicted physicians found that parental deprivation and prolonged or childhood illness were statistically important determinants [J*Hall, Stickney, Popkin, 1978]. A profile of the physician narcotic addict suggests that the following all constitute high-risk factors:

1. childhood illness;
2. psychopathology in the family (even though the family was physically intact);
3. fathers with serious drinking problems;
4. intense feelings of dependency and resentment towards their mothers [J*Anonymous, 1972b].

The "Precursors Study" done at Johns Hopkins was a long-term prospective investigation of 1,337 medical students registered in classes graduating during the years 1948 through 1964. It examined drinking by a cohort of male graduates and found that

"when youthful personality characteristics of midlife groups were compared, midlife drinkers were found to have been more anxious under stress than midlife non-drinkers." Additionally, this study found that those who would later become ill could be identified by their reaction to stress during medical school:

1. they manifested more anxiety and anger;
2. they had more difficulty sleeping;
3. they used both tobacco and alcohol more frequently.

These reactions to stressors during medical school seem to represent predictors of both later physiological and psychological morbidity [J*Thomas, 1976; J*Thomas, Luber & Smith, 1977; J*Thomas, Samtara, & Shaffer, 1980].

There are some reasons to believe that early childhood experiences may influence some people to choose a career in medicine. Some people may seek to gain mastery over suffering and sickness by becoming physicians, and they may be motivated to heal others in the hope that they too may be healed.

What are some of the other factors that may lead to a person's decision to become a physician? Cochrane [B*Coombs & Vincent, 1971] writes that important responsibilities, self-determination (rather than subordination), the chance to help others, prestige, and financial security characterize the physician's life. "Many persons who select a career in medicine are hard working and goal directed, characteristics which are rewarded during professional training but may lead to disillusionment in the less structured clinical practice years" [J*Shortt, 1979]. The result may be a maladaptive response to the stress of practice. Additional evidence exists that chemically dependent physicians are often in the top part of their class academically (overachievers) and that they often did not really want to be physicians (they may have succumbed to parental influence) [B*Whitfield, Bissell & Wesson, in press].

Medical training is long and arduous, and it creates stress for all who undergo it. Inadequate sleep, poor dietary habits, lack of regular exercise, and unfulfilling intimate relationships are some of the stresses during this period [J*Loes & Scheiber, 1981; J*McCue, 1982]. Psychological stress can be caused by prolonged emotional and financial dependency, by having to postpone tangible evidence of becoming a physician, by impaired self-esteem, and by the initial encounter with death. Physicians who fail to achieve a consonance between their expectations of themselves (and others) and what they find in reality may resort to immature or neurotic defense mechanisms [B*Vaillant, 1977]. Physicians are not only prone to overwork, but they are trained (or brainwashed) to accept unreasonable work conditions as necessary for competence or as the cost of being "special" [B*Gerber, 1983]. It is not enough to work 60-80 hours a week at clinical duties, but physicians also must spend additional hours reading journals to keep up with new discoveries.

Do career choice and role strains put the family physician at higher risk? Physicians engaged in direct patient care, and certainly family physicians, experience more role stress than other physicians who relate less directly or continuously to their patients. Many clinical cases present in undifferentiated ways, leaving considerable uncertainty about diagnosis and treatment. Family medicine is a relatively new specialty, producing ample opportunity for family physicians to suffer some self-doubts about professional identity. We are still called upon by the older specialties to defend our legitimacy as a specialty. The lower level of financial reimbursement to family physicians, who are clinically qualified to perform a particular procedure or provide special care similar to other specialists, is slowly disappearing, but it is still frustrating. In academic institutions, family medicine is still fighting for equal stature with the other academic departments. The first contact role of the primary care physician invites frequent disruptions of personal life if continuity of care is maintained. The family of the physician fail to accept the justification for repeated disruptions of their dinners, play times, and even sexual intercourse without at least some complaints [B*Gerber, 1983].

PREVENTION AND TREATMENT

What are the AAFP, the STFM, and the family practice residency training programs providing in the way of prevention and support for their members? To cover all the existing programs that address the problem of physician impairment is beyond the scope of this monograph. Our focus will be on those programs designed for learners in undergraduate, graduate, and postgraduate family practice or family medicine.

Primary prevention acknowledges the fact that a large population is at risk because of exposure to a disease. Being a physician may indeed "be hazardous to your health," both emotionally and physically, and an increasing number of medical schools are beginning to address this fact. Some programs present both lectures about the impaired physician and opportunities to interact with physicians who are recovering from alcoholism and other addictive disorders. However, few have employee assistance programs (EAPs) at their medical centers. Brown University has a Student Assistance Program and offers counseling for children of alcoholic parents [R*Brown].

Many of the family practice residency training programs have addressed the problems of resident impairment by developing a network of support systems that provide didactic information, such as preventive counseling for faculty and peers, spouse/partner group activities, and by such measures as values clarification and encounter or sensitivity training. Free brief psychotherapy is offered at UCLA [J*Borenstein & Cook, 1982]. Family therapy is often helpful in dealing with impairment [B*Knutsen, 1978]. Many family medicine residencies employ the Balint technique of discussion and support around difficult clinical situations which arise in practice [B*Balint, 1957].

In 1971, the AAFP's Committee on Mental Health directed its attention to the mental and emotional well-being of its members. Since that time, it has presented many programs that have, as their focus, the stressors of active family practice and the development of healthy coping mechanisms. These programs have included spouses and in some instances the children as well.

Those who are at high risk for impairment can be recognized by the preprofessional stresses already defined, and by early indications of difficulties in coping with the demands of training. At probably the highest risk for becoming chemically dependent are the children and grandchildren of chemical dependent people. Because most victims cannot recognize their relatives as such, it would help to ask, "Have I ever been concerned about any of my relative's drinking or drug use?" If the answer is yes, one can answer the questions on the Michigan Alcoholism Screening Test [J*Selzer, 1971] as though the relative were answering honestly. In a survey of family practice residents at the University of Maryland, Whitfield found that 35% had an alcoholic parent.

Early case identification should be the responsibility, or at least the concern, of alert and sensitive faculty and peers. Unfortunately, until quite recently, faculty and peer identification and confrontation were not included in the concept of "being one's brother's keeper." Support, via counseling, by those who are qualified to give it, and with due respect to confidentiality, should be readily accessible. Family practice resident organizations have developed the D.O.C. [R*Doctors Ought to Care] program, which aims at developing a healthy life-style (proper nutrition, regular exercise, moderate use or the avoidance of all chemicals, etc.) not only for patients, but for doctors themselves.

Most secondary and tertiary prevention (remedial intervention and rehabilitation) is modeled after the [R*AMA] programs, consisting of early case identification with constructive, supportive confrontation offering specific remedial aid. These programs had their initial impetus when concerned professional organizations, medical educators, and practitioners (who had first-hand experiences with physician impairment) were sufficiently alarmed about the morbidity to take positive action.

The loss of physicians from an active, productive professional life and the cost, both economic and emotional, of CD are cause for genuine concern. Those of us who are knowledgeable about the early signs and symptoms of impairment and chemical dependence can and should serve as early case-finders and collaborate with others in confrontation and intervention efforts. Of the roughly 900 physicians studied in 16 published studies, 63% seemed to do well [J*Vogtsberger, 1984]. There is evidence that a high percentage of physicians who commit themselves to a sound treatment program with an adequate ongoing follow-up can make a strong recovery and once again practice their profession unimpaired. They can also enjoy the experience of spiritual growth [B*Whitfield, 1983] and can carry their newly acquired

understanding of themselves and their recovery into their professional role by helping to prevent or rehabilitate CD in their patients. One can locate local resources for dealing with physician impairment by contacting the local or state medical society or checking with the hospital employee assistance program. One should become familiar with the philosophy and approach of local programs (whether coercive or voluntary in nature) before using the program. However, it is important to avoid enabling the physician to elude appropriate treatment because of the need to maintain the practice or finish a training program [J*Carlson, 1984].

PHYSICIAN IMPAIRMENT

LEARNING OBJECTIVE: Physician impairment

The resident should become knowledgeable about the epidemiology of physician impairment, approaches used by prevention and intervention programs and recovery rates.

TEACHING HINTS: Physician impairment

Residents could split up the reading assignments and lead a seminar covering the facts about physician impairment and its prevention. Or they could meet with several recovering CD medical families to discuss the experience [R*IDAA].

EVALUATION: Physician impairment

A. The resident should be able to estimate the prevalence of CD among physicians, which runs at about 10%, with a range as high as 18%.

B. The resident should be able to list at least four causes of chronic impairment among physicians, and give an estimated percentage for each.

1. Alcoholism and other CD: 85%
2. Mental illness: 5-10%
3. Senility: 2%
4. Physical illness: 2%
5. Miscellaneous: 3-5%

C. The resident should be able to briefly define the term "impairment" as it applies to the physician.

ANSWER: A physician can be considered to be impaired when one or more problems cause him or her to be recurrently troubled or dysfunctional in the quality of patient care, other professional activities, education, or private life. Impairment in the professional area may be further defined

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as a physician's trouble with personal difficulties to the extent that he or she cannot:

1. offer reasonable patient care;
2. effectively promote health through interpersonal skills;
3. maintain skills by continuing education;
4. maintain a prior level of functioning, even if currently functioning adequately.

Impairment may be further characterized by denial and ambivalence on the part of the physician, the family, colleagues, and the community, all of whom may ignore the problem until it is too late. Intervention with appropriate treatment as soon as impairment is recognized is a major goal of the impaired-physician movement. Impairment may be either transient or chronic, and varies along a spectrum of severity of degree and causes.

D. The resident should be able to list at least two symptoms or signs of physician impairment in each of the following areas:

1. home and family;
2. office and practice;
3. hospital;
4. friends and community;
5. physical health;
6. employment applications. (See [B*Whitfield, Bissel & Wesson, in press] for answers.)

E. The resident should be able to briefly describe or list at least three characteristics that may be associated with higher risk of physician chemical dependence.

1. alcoholism in parent (or a rigidly abstaining parent and an alcoholic grandparent);
2. feeling forced into medicine;
3. academic standing in the top portion of his or her medical school class;
4. heavy use of alcohol or drugs;
5. smoke cigarettes;
6. male gender [J*Wallot & Lambert, 1984].

The resident should understand that these are only associations, and thence that they do not absolutely predict, and conversely that their absence does not protect one from developing, addictive disorders.

F. The resident should be able to estimate the success rate of treatment of chemical dependent physicians when managed through an appropriate state medical society rehabilitation program.

ANSWER: The success rate stands at about 70-83%.

SELF-AWARENESS OF RISK

BEHAVIORAL OBJECTIVE: Self-awareness of risk

The resident will be sufficiently aware of the personal vulnerability of developing problems to afford a realistic degree of self-examination and monitoring, and to promote an openness to expression of concerns by peers and superiors.

TEACHING HINTS: Self-awareness of risk

The major hurdle to be traversed is to get beyond personal denial of vulnerability. A group of residents could meet with several formerly impaired physicians and their family members to discuss the factors that led them to have problems. They might also inquire about the ways in which these doctors and families initially resisted change, were convinced to accept help, and then recovered.

The residents might watch films [A*Gallant: Alcohol and Drug Abuse among Physicians; A*Don't be scared; A*Our Brother's Keeper] or read [B*Gerber, 1983; B*Shem, 1978; B*Twerski, 1982] before discussing personal vulnerability and stress during training with one another in a small group (with or without their significant others present).

The residents could list and discuss personal risk factors for potential impairment according to the following headings:

1. family history;
2. alcohol or other drug use;
3. diet, sleep and exercise;
4. overwork, recreation and relaxation;
5. regular ventilation of frustrations to a trusted friend;
6. stress coping styles;
7. stress exposure.

Then they might discuss alternatives to their current life-styles that might provide greater protection to one another. The resident could write a plan to lower personal risk regarding each of the areas listed above. One could also describe the special pressures imposed on the family of the family physician. The resident could write a plan to respond to each of these pressures.

Residents might pair up to do analyses of each other's risk factors, and counsel one another as they might counsel a patient. After completing one round, they might switch roles so as to minimize the inequity of roles. They could analyse the special stresses they have experienced during their training and develop a plan for dealing with those aspects that can be altered.

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Spouses might be assembled to discuss stress during training. They could use [B*Anonymous, 1982] as a guide for their discussion. They could then meet with the resident group after having met with themselves for awhile, to present their concerns and further discuss what might be done about them.

EVALUATION: Self-awareness of risk

Several times during the residency (e.g., annually) have each resident do a risk assessment with records kept of previous assessments and plans, to evaluate the accuracy of predictions and to assess the effectiveness of prior plans.

A CONSTRUCTIVE APPROACH

BEHAVIORAL OBJECTIVE: Constructive approach

It is important for the resident to take a constructive approach to a troubled colleague just like one would to a troubled patient in one's practice. The resident should be able to demonstrate the skill at confronting the impaired colleague or responding effectively to the situation.

TEACHING HINTS: Constructive approach

One might set up a seminar psychodrama in which several residents are aware of a troubled peer or superior or student. They can spontaneously play out their reactions to the problem to others in the group, who may periodically stop the psychodrama to interject feedback or to discuss the ethical and pragmatic dilemmas which arise. The following schema might be used to structure various scenes in the psychodrama:

1. substantiation that the observations are true;
2. contacting the state medical society or other state professional society's rehabilitation or impaired physician committee;
3. description of observations and concerns, and identification of the professional in question;
4. providing the names of two other people who can substantiate the observations;
5. offering help to the colleague individually or through such a committee;
6. recontacting the committee to help it to monitor its effectiveness.

If it is possible to arrange with the state or local medical society, interested residents might be permitted to attend a meeting of the impaired physician committee or accompany an intervention team.

CHAPTER 9: FAMILY PHYSICIAN AT RISK

Unfortunately for us all, there are usually many examples of resident impairment, medical student impairment and even faculty impairment around us. Actually dealing with one such situation, and including the residents in the action, may provide the best "field experience" one could get. But, beware of the situation where the residents challenge faculty resistance to taking a constructive approach with a colleague. Failure to follow through in a real life situation may teach the residents to do as we do, rather than as we say.

EVALUATION: Constructive approach

The subjective observation of the psychodrama approach may be used to evaluate the degree to which the resident has internalized the principles of a constructive approach. In the psychodrama, watch for enabling activities and reluctance to take a "tough love" constructive approach.

However, a psychodrama is never as good as real life. The resistance to taking a constructive approach when the target impaired colleague is a close friend, partner or relative cannot be duplicated in an artificial situation of a psychodrama. Asking the resident about situations in which the constructive approach has been used to help impaired colleagues, or in which impairment was encountered but nothing was done, may help to evaluate the progress of the resident over time.

CHAPTER 10: STRATEGIES FOR CURRICULAR CHANGE

By Ardis K. Davis, Peter G. Coggan & Michael R. Liepman

In formulating an approach to the development of a substance abuse curriculum for family medicine, it is important to consider first the overall strategies at broad institutional and community levels before the specific design of the curriculum is undertaken. The comprehensiveness, depth, and longevity of the overall effort will depend largely upon careful consideration of many strategic approaches. The purpose of this chapter is to outline strategies that will help ensure that substance abuse training programs will be successful. The initial decisions to be made revolve around how comprehensive the curriculum efforts should be, and how long-lasting an impact they are intended to have. For some departments of family medicine, the expectations may be higher than for others. This outline and discussion should cover all the strategic bases to ensure that curriculum goals in substance abuse will be best attained.

GUIDELINES TO CURRICULAR CHANGE

Five guidelines for successful substance abuse curriculum development for family medicine follow:

INTERDISCIPLINARY APPROACH

Alcoholism and other substance abuse are so pervasive in their impact on individual, family and societal functions that one cannot possibly teach, in family medicine, all there is to know about these problems. Substance abuse crosses all biomedical and psychosocial boundaries, and thus demands an interdisciplinary approach to teaching. In the past, comprehensive management of substance abuse has not been within the province of medicine, but rather has been scattered over other health-care disciplines. Involvement of these disciplines is essential in formulating a curriculum plan.

TEAM APPROACH

Because of the interdisciplinary nature of substance abuse treatment, it is important to emphasize how various health-care professionals can manage the problem together. Students and residents in family medicine should learn to appreciate the complexity in the management of chronic alcoholism and other substance abuse. They should learn that physicians cannot, and should not be expected to, manage the problem alone. An awareness of how specialized programs and family physicians can complement one another in identifying and rehabilitating victims of the addictive disorders is essential to understanding the scope and limits of the family physician's role.

INTERFACE WITH COMMUNITY

The necessity for emphasizing the team approach in substance abuse management demands an interface between the residency program and treatment resources in the community. Clinical teaching about substance abuse must take place where the care takes place. Community treatment programs, both medical and non-medical, plus self-help groups, are all essential elements of a successful clinical treatment repertoire. Residents cannot comprehend the care for substance abusers without intimate knowledge of such resources. Substance abuse itself is a community-based phenomenon: bars, dope dens, package stores, pushers, country clubs, flop houses, parties, emergency rooms, scrip docs, and narcs. Each play a role in the lives of some substance abusing patients. In a sense, these are part of the patients' social network. Likewise, the families and work world, which often encourage the use of psychoactive substances, are also a part of the patients' community. Secluding the residents in the "ivory tower" of their hospitals and clinics gives them an unrealistic view of their patients' world. Yet, it is also necessary to help the residents to see how substance abuse presents in the medical world, and how the family physician can interface between the patient, the community and the hospital or clinic [J*Clifford, 1959].

BLENDING BASIC SCIENCE WITH CLINICAL KNOWLEDGE

An understanding of substance abuse requires knowledge from the basic sciences in order for physicians to function well clinically. Thus, the teaching must blend basic science with clinical. Failure to learn the basic science leaves the physician no more advanced than the lay alcoholism or drug counselor, while failure to learn the clinical leaves the physician indistinguishable from the laboratory scientist.

SUBSTANCE ABUSE AS A PROTOTYPE

Few chronic illnesses provide a better opportunity than substance abuse to illustrate the breadth of family medicine's basic principles. Family medicine's foundation lies on the comprehensive management of several common problems, emphasizing the interrelationship among biomedical, psychosocial, and socioeconomic factors, and their impact on disease management and prevention. An example of how the goals of an alcoholism curriculum can be tied into the principles of family medicine is presented in Table 10-1. Use of substance abuse as a family medicine prototypic illness can be useful in gaining support from colleagues for inclusion of substance abuse training in the mainstream of the residency curriculum. Otherwise, there is a risk that it will be relegated to the status of a subspecialty topic, deserving of very little attention.

TABLE 10-1: Alcoholism as a Prototypic Biopsychosocial Disease
Comparison of Alcoholism Teaching Objectives
with Family Medicine Principles

FAMILY MEDICINE PRINCIPLES	ALCOHOLISM TEACHING OBJECTIVES
Comprehensive care, disease prevention, health promotion	Risk identification Early recognition
Commitment to care of persons and families in community context	Identification through family and community, family support, confrontation and intervention
Commitment to family physician as member of health-care team	Referral to community resources and team approach to management
Provision of continuity of care	Outpatient management & follow-up
Concern for the subjective aspects of medicine	Physicians at high-risk of impairment

COMMENTS: STRATEGIES FOR INSTITUTIONAL CHANGE

In family medicine, there are various levels (from the broad to the specific) that can be considered as targets of strategies for curriculum development in substance abuse. Some are institutions connected with medical care and education, others are portions of the non-medical community.

THE UNIVERSITY

When the residency training program is part of or affiliated with a university, there may be contributions of other disciplines and departments within the university that pertain to substance abuse (e.g., pharmacology, pharmacy, toxicology, nursing, social work, public health, counseling, education, psychology, to name a few). The presence of a department does not guarantee that persons with expertise and/or interest in substance abuse will be found. But key faculty from a variety of related disciplines and departments should be consulted early in the formulation of a curriculum plan if they are eventually going to be supporting the movement to improve and maintain a substance abuse training program. This consultation is important for three reasons: (1) it ensures an interdisciplinary approach to teaching; (2) it simplifies significantly the decisions on the most current, important information to be conveyed about alcoholism and substance abuse; and (3) it emphasizes an awareness for medi-

cal school faculty, residents, and students that medicine is not expected to know about or manage all facets of the problem. A side benefit of such interdisciplinary, interdepartmental consultation may be the development of substance abuse training programs for a variety of other trainees from other disciplines.

THE MEDICAL SCHOOL

Including the medical school dean's office in discussions and decisions early in the planning may be a crucial step in ensuring a lasting impact of undergraduate curriculum development efforts on the medical school. Without the authority and sanction granted by the dean's office, efforts may soon die once key persons at lower administrative levels cease their activity in this area. The process of negotiation at this level may take time and may require compromise, but the end result may well be worth these inconveniences.

It may be necessary to appeal to the competitive side of medical school administrators to get their cooperation. Educating them about recent developments in substance abuse medical education may help [J*AMA CMH & CADD, 1972; B*Pokorny, Putnam & Fryer, 1980; J*Solomon & Davis, 1978]. Results of the school's own medical students' scores on substance abuse questions on the National Board exams may help to point out deficiencies in teaching [B*Griffin et al., 1980]. The overall breadth and depth of substance abuse teaching programs throughout the medical school may be assessed in contrast to other medical schools [B*Pokorny, Putnam & Fryer, 1980; J*Solomon & Davis, 1978].

Identification of key faculty members with needed interest or expertise from other medical school departments, and their early involvement in curriculum planning, is helpful for six reasons: (1) as with other university faculty members, they can provide much information expediently; (2) many departments have specific bodies of knowledge that should be drawn upon for comprehensive teaching in family medicine, such as psychiatry and pharmacology; (3) duplication of teaching can be avoided to help ensure an efficient and well-rounded curriculum for students across departments; (4) there may be specific rotations or experiences available through other departments that could augment curriculum in family medicine; (5) the credibility of efforts in family medicine will be strengthened by receiving approval and input from consultants from other departments, especially if the eventual product is consistent across departments; and (6) these consultants may be influential with the curriculum committee. While most of this discussion is germane only to medical student curriculum, the creation of clinically-based training rotations within a medical center that focus on substance abuse may be very difficult without a broad base of support.

In non-academic residency training programs, the hospital may play a significant role in determining curricular matters. Again, help from other departments is important in gaining support and access to resources.

THE FAMILY MEDICINE DEPARTMENT

At the departmental level, there are a number of important strategies to consider. First, identification of any faculty members with expertise or interest in this area is important. Regardless of how many faculty members may have interest or expertise, it is vital that one faculty member be given primary responsibility for overseeing the curriculum development program. However, this need not be someone with expertise. Interest and enthusiasm for teaching this topic are the paramount characteristics that may be employed to direct all the efforts and coordinate other departmental, interdepartmental, and university faculty members with expertise. This person should also maintain open communication about the curriculum activities between him- or herself and the department chairperson, as well as with the different sections of the department, and with the medical school administrator. All of these administrative steps help to ensure that the department as a whole has some commitment to the program, and not just one individual. This has important implications for the longevity of a substance abuse curriculum in the department and the school.

In many departments of family medicine there may be few, if any, faculty members with knowledge, skill or interest in the area of substance abuse [J*Fisher et al., 1975 & 1976]. Therefore, there are few role models for students or residents. Faculty development is perhaps the most vital strategy at the departmental level for two reasons: (1) much of what is taught in family medicine is on a clinical level; if there are no faculty members with clinical skills in this area, students and residents will have no role models and, thus, far fewer opportunities to develop skills; and (2) a curriculum program in substance abuse cannot survive without an interested and knowledgeable faculty. A single faculty member may provide the initial momentum, but certainly cannot carry the ball forever. This problem manifests itself at other levels as well, so having interdepartmental and university wide involvement and support only strengthens the likelihood that an effort will thrive.

The following questions must be considered before embarking on a faculty development program: What are the needs of the faculty? Is there a deficiency in knowledge or attitudes or both? Do faculty members perceive their deficiencies? How many faculty members should receive training, and in what depth? Should all faculty members receive minimum training, or should a few receive in-depth training? How should training be accomplished, and who should do it? How will the training be done, and who will make faculty time available for this faculty development activity? These questions dictate the course of faculty development activity. Some useful faculty development strategies are listed below:

1. A knowledge/attitude survey can be conducted to ascertain strengths and deficiencies of faculty in clinical care of patients and families with substance abuse problems [J*Liepman, Whitfield & Landeen; J*Mueller &

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Ferneau, 1971) which might help or impede teaching about substance abuse care.

2. If possible, it is very useful to have local family physicians with substance abuse expertise participate in the training. An interdisciplinary approach is realistic, but an illustration of the family physician's role is extremely helpful in stimulating interest and credibility among faculty members.
3. Given the time constraints of most faculty members, a half-day workshop may be the ideal length of time. Examples of a faculty development workshop structure that touches the relevant topics for family physicians are available in [J*Brennon, Dixon & Snider, 1974; B*Jackson, 1980; J*Liepman & Gilbert, 1979] and in TABLE 10-2. This format has also been useful for residency teaching.
4. Perhaps the most useful approach to the training of faculty is the provision of opportunities for clinical experience in a specialized treatment program. This is probably the most influential mechanism for changing stereotypical attitudes, and it can be provided as either a simple site visit, or expanded into a mini-residency. Most treatment programs (in our experience) are delighted to accommodate needs such as these. Attending an AA meeting with a credible sponsor (such as a recovering alcoholic or substance abusing physician) is another experience that may enlighten the faculty on the place of AA in sobriety maintenance.

In addition to these direct approaches, there are some useful indirect strategies to arouse faculty interest. The prevalence of substance abuse problems in local practices or in hospital populations is often documented. Presenting this information to faculty may stimulate interest and motivation to learn more about this area. Another indirect strategy is to teach students and residents about substance abuse so that they ask other faculty members questions. In this way faculty can become aware of their own knowledge deficiencies, and may even learn from the trainees. If they do not become defensive, they may seek supplemental training in order to teach better about substance abuse. However, if they become defensive, they might undermine the teaching which is being undertaken. This indirect approach is more acceptable when there is gradual implementation of curriculum changes in the training program.

TABLE 10-2: Sample Short Alcoholism Faculty Workshop

TIME	WORKSHOP FORMAT	WORKSHOP CONTENT
10 min.	Slides	Prevalence and magnitude
40 min.	25 min. trigger film [A*Enabler] and discussion	Attitudes towards the family illness concept
30 min.	Slides and videotaped clinical interview [A*Liepmann]	Diagnosis via history, lab, confrontation, & physical exam
10 min.	B R E A K	Alternative beverages, fruit
30 min.	Small group role-play: 1 facilitator per group	History-taking and supportive confrontation
30 min.	30 min. film [A*The Intervention]	Encouraging treatment via family & peer pressure
15 min.	Lecture and discussion	Outpatient withdrawal
15 min.	Lecture and discussion	Local community resources

COMMUNITY LIAISON

The interface between the community and institution is vital to the success of a substance abuse curriculum development effort. Clinical teaching sites in the community must be carefully selected to optimize the educational value of a block rotation or a visit. The teacher should meet and work with them to explain the objectives of the time spent there, to prepare the agency staff for the needs and assets of the residents or students, to decide upon the most appropriate activities and schedules, to obtain evaluative feedback from the agency about trainee performance, interest, behavior and attitudes, and the agency reaction to their participation in the training. Invariably, first attempts will need improvement, and an active liaison between the family medicine department and the agency will facilitate rapid adjustments early in the team effort. In addition, taking some time and effort to augment public relations will strengthen the long-term success of a program. These two strategies are discussed below.

COMMUNITY RESOURCES

In approaching community resources and programs, it is important to consider that there are many biases about how substance abuse is best treated. Physicians as a group also have a poor reputation in the community of recovering victims and their counselors because of their general ignorance about the subject and their tendency to misdiagnose and mismanage the condition. This skepticism and these biases should be discussed openly with community members in the preliminary planning phases. The participation of community treatment programs and other recovery aids such as the self-help groups in physician training can be explained as an opportunity to rectify past short-comings in physician education about substance abuse. It is important that these controversial issues be discussed prior to trainee arrival at the program. While it is not harmful for them to learn of the bad reputation doctors have, they must be given a fair opportunity to earn a good reputation by being interested in learning all about substance abuse care. The particular biases about treatment modalities (e.g., nobody can recover without AA, methadone maintenance keeps you from using drugs, methadone is just another drug) may motivate certain substance abuse professionals to behave clinically in a stereotyped manner with which professionals in another agency might disagree. Trainees will need to be warned of these biases and preferences beforehand so that they do not challenge the status quo in these agencies too aggressively.

The first question to answer is what learning objectives might be achieved through the direct involvement of community resources in teaching. Trainees can learn about the variety of treatment approaches that can be used, the most appropriate use of community resources in clinical management of a case, the importance of respectful collaboration between the doctor and the counselor, a realistic attitude towards treatment outcome, and the need for persistent motivation towards recovery. They might also be introduced to the manipulateness of certain clients and observe how counselors defeat the con games. In particular, they might interview clients to determine what interaction they have had with the medical sector, and what their experience was like. In addition, the trainees might survey clients to determine the nature of their involvement in treatment over time, focussing on transitions from active illness to active recovery and back.

The teaching objectives of the module must be linked with the available time for clinical community site experiences. If there is sufficient time to visit several programs, this should be encouraged and arranged. If not, the programs might be invited to supply some pamphlets describing their program or present a poster or slide presentation at a didactic session. Care should be taken in the selection of panel members and in the prepresentation briefing. Strong biases or overstated opinions will destroy the credibility of a panel, and may do irreparable harm. If there is ample time for extended clinical field placements, in-depth clinical rotations may be arranged at selected agencies.

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The number and types of programs available in a community will determine how community resources might be used. The local or regional alcohol information center should be able to provide information on the programs in a particular area. In most large communities, there is a great variety of outpatient and inpatient programs and centers. Smaller communities are often not so well endowed, but some resources will frequently be available in virtually any community (e.g., AA and Alanon). The willingness of staff in community programs to become involved in teaching of medical students is another important consideration. This can be easily assessed through visits and discussions with staff. In some cases there will be resistance to the intrusion of the students into the inpatient program milieu or the group process. Initial fears are generally swept away if interested trainees behave respectfully towards staff and clients.

The geographic distance from the residency program to community agencies may determine at least in part how accessible they are for teaching purposes. Students and residents have been allowed to spend time "in-house" at many programs in the Seattle area--this might be an option if clinical teaching time is available and if a desired program is far away from the clinic or classroom.

The level of student or resident involvement in community programs will be determined by the objectives, the amount of clinical teaching time, and agency limits. The curriculum planners may have to decide whether students or residents should make only brief, half-day visits to one or more programs, or if they should spend extended time in clinical rotations at one program. Potential student and resident involvement might range from observing for one or two visits, to participation in patient care and program activities. The types of student and resident activities desired will have to be negotiated early with program personnel. It has been our experience that most programs are very flexible, and that the staff are happy to have students and residents become actively involved.

Due to the variety of treatment approaches and philosophies, the particular approach used in a community program may not coincide with the objectives of the teaching module, especially if only one community site visit or rotation are planned. Exposure to several programs on a superficial level may be advantageous in the breadth and potential success of different treatments and philosophies.

Many treatment programs include outpatient and inpatient components, ranging from detoxification to group therapy to drunk-driver classes to biofeedback to relaxation training to family counseling sessions. The number and diversity of components in a program, as well as the potential for student and resident involvement in each, will help in decisions regarding the choice of program, as well as in the structure of clinical time in the community site. Each potential site should be evaluated in terms of the appropriateness of what is taught to the training needs and baseline assets of the resident.

Community treatment programs vary considerably according to the level of medical supervision and the use of allied and paraprofessional personnel. They also vary in terms of their experience with health profession students and residents rotating through their programs. Staff familiar with teaching, educational philosophy, and the expectations of students and residents may facilitate the smooth integration of students and residents into their programs. Staff unfamiliar with teaching may need preparatory guidance on the expectations of the clerkship and student and resident roles, with perhaps closer consultation with medical school faculty in the initial phases.

PUBLIC RELATIONS

There are some useful steps that can be taken to achieve good public relations, which in turn enhance the success of a program. Such strategies are (1) offering continuing medical education workshops to practicing family physicians in the community; (2) joining local medical society committees on alcoholism and substance abuse, or any other local community committee efforts in this field; (3) utilizing community treatment representatives on an advisory panel for planning, as discussed earlier; and (4) being careful not to buy into any mainstream biases regarding treatment philosophies, in order to protect against political ramifications within the community. The field of alcoholism and substance abuse treatment is politically quite volatile. Maintaining an open mind towards all parties is the most diplomatic strategy. By making connections with the substance abuse community, one will attract resources and gain a community credibility. Members of the community who first greet the effort with suspicion may eventually judge it kindly if it appears to be making a contribution to solving the problem of medical professional ignorance about substance abuse care. By participating in the community activities, one will learn of available resources, opportunities, needs and projects. This information may be invaluable when planning curricular offerings in the community. Your expertise and that of trainees in the department may be tapped in ways that were never anticipated, for the mutual benefit of the community and the department (and its curriculum).

LEARNING OBJECTIVES: Resident Training

Table 10-3 lists a series of learning objectives for family medicine residents. Each residency training program may wish to select some of these as minimal required objectives while identifying others as valuable elective objectives for interested residents. Details about many of these objectives may be found elsewhere in this monograph.

TABLE 10-3: SUBSTANCE ABUSE LEARNING OBJECTIVES

	KNOWLEDGE	ATTITUDE	BEHAVIOR
SUBSTANCE USE TYPOLOGY	Distinguish between abstinence, use, abuse, dependence	Respect each kind of user or abstainer; Examine attitudes	Gather pharmacologic history & diagnose problematic use
DRUG CLASS TYPOLOGY	Know psychoactive drug class & action, dosages, routes of administration, body distribution, cross-tolerance & dependence potential, elimination kinetics	Respect people who abstain, use, abuse drugs in each class and examine own & ethno-cultural societal attitudes	Gather historical detail about route, action, dependence & use to anticipate problems from use, abstinence, and elimination kinetics
EPIDEMIOLOGY	Know prevalence of each type of use of each drug class & high-risk factors	Examine stereotypes of persons in each usage category & contrast with facts	Use screening tools to identify cases & contrast prevalence estimate vs. # found
ETIOLOGY & CONCEPTUAL MODELS	Know etiologic theories & evidence on which each model is based	Examine own beliefs in chronic disease model & views about etiology	Explain disease concept to patient & family
ACUTE & CHRONIC CONSEQUENCES OF ABUSE OF DRUGS	Know common social, psychological & biological sequelae of drug abuse or sudden cessation; understand defensive denial	Empathize with the victims of damaging consequences of substance abuse or abstinence; contrast own health concerns vs. patient denial	Use medical review of systems, physical exam & lab tests to identify sequelae; warn victims of hazards in continuing to abuse
ACUTE INTOXICATION & OVERDOSE	Know causes of toxic delirium; signs of drug synergy & OD; antidotes & support of vital functions; methods to identify drugs & hasten their elimination	Examine own attitude towards intoxicated person & affective reaction to person out of control of sensorium, behavior, & bodily functions	Qualitatively & quantitatively evaluate intoxicated patient for delirium by history, physical & lab tests; calm intoxicated person, manage overdose
BENEFICIAL ACTIONS OF PSYCHOACTIVE DRUGS	Know benefits of drug use & losses of those who must quit taking them	Examine own feelings about abstinence & drug use; empathize with those who quit	Nonjudgmentally ask patient & family benefit of drugs & impact of quitting

TABLE 10-3: SUBSTANCE ABUSE LEARNING OBJECTIVES (continued)

	KNOWLEDGE	ATTITUDE	BEHAVIOR
CHEMICAL DEPENDENCY IN FAMILIES	Know that family behavior changes when abuser uses; understand enabling & family roles	Examine impact on own family of abuse of drugs by relative & own attitude towards enablers	Gather family history watching for roles, enablers, & behavioral shifts with drug use
BEHAVIORAL CONTROL & REINFORCERS	Understand motives for use as opposite of non-compliance	Examine own use of drugs & compliance to healthy behaviors	Realistically advise healthy behavior & reinforce success
MOTIVATION TO RECOVER & COERCIVE TECHNIQUES	Know ways to coerce patient to accept recovery early; understand defensive resistance of change	Examine own reaction towards patients and families who are reluctant to change; ethics of neglect versus coercion	Analyze motivational status of patient & family; utilize coercive resources & opportunities to motivate change
DETOXICATION	Know signs+symptoms of acute withdrawal to each drug class & treatment	Examine own attitude towards withdrawing patient & impact it has on care offered	Medically support withdrawal in clinic or hospital; suggest rehabilitation
TREATMENT & REHAB	Know treatment modalities and local resources; understand outcomes	Examine expectations of treatment & frustration over relapse	Gather history of prior treatment & outcome; evaluate causes of relapses
REFERRAL & TEAMWORK WITH LOCAL RESOURCES	Know treatment philosophy & methods where patient was referred	Examine own feelings about treatment & impact this has on cooperation offered	Match patient needs to program resources & monitor progress & communicate often
CAUTIOUS PRESCRIBING PRACTICES	Know risks of inducing relapse by prescribing abuseable drugs to recovering patients	Examine own wish to rescue patient from discomfort & choice to prescribe drugs, hence enable relapse	Warn patient of addiction risk from drugs & offer non- drug alternatives for symptoms
PHYSICIAN IMPAIRMENT	Know risk & local constructive remedies for an impaired colleague	Examine reluctance to confront impaired colleague & need to protect patients	Confront colleague with concerns or initiate steps to motivate recovery

COMMENTS: Resident training

The training of family medicine residents about substance abuse can be done in several different ways, depending on teaching resources and the format of training in the residency program. The following questions and accompanying discussion may help clarify how the training options may be matched to the needs and assets of the program.

Is a longitudinal approach, with integrated teaching components in all three years of a residency training program better suited to a residency program than a modular or block rotation approach, with one intensive alcoholism teaching experience in the third year of training? The most integrated approach is to develop a curriculum that spans the length of the three-year residency. This is more complex than the block rotation approach but, if feasible, it allows for a more comprehensive curriculum that builds on itself over the three years. With such a strategy, didactic seminars and workshops can be integrated into the existing seminar series for each year. Clinical experiences can be incorporated into the second and third years--for example, site visits during year two and a mini-rotation in a substance abuse treatment program in year three. Another option allows residents to follow alcoholic or substance abusing patients and their families from among their practice throughout the three years of the residency, providing continuous and comprehensive care. However, the block rotation in the second or third year may be the most practical alternative for many residency programs. If the experts on substance abuse are remote geographically, patients will be sent far away for specialized care. Likewise, block rotations will be done away from the home base. If no one on the faculty feels competent to provide rehabilitative care for alcoholic patients, there will be no role models to learn from during an integrated training program.

TEACHING HINTS: Resident training

CONTENT

The options relevant to family medicine include the following: physicians' attitudes; risk assessment and prevention; early diagnosis, emphasizing interviewing skills and recognizing signs and symptoms; treatment planning; detoxification; referral to and cooperation with community resources; supportive confrontation and motivation; supporting the family; monitoring progress of recovery and relapse management; and recognizing and responding to physician colleagues who are at risk for substance abuse. (More details about specific content areas may be found elsewhere in this monograph.)

CLINICAL FORMATS

Clinical experience might be gained by required supervised longitudinal ambulatory management of a specified number of families suffering from substance abuse problems from within training practices. The advantage of this approach is that it teaches the residents in a setting that most closely resembles the real world. Residents will have the opportunity to encounter the problems that are likely to occur during post-residency practice, while still having supervisors around to help in their resolution. This approach also provides a chance to watch the evolution of the recovery or the progression of the unarrested disease longitudinally. The major drawback to this approach is when the residency program is unable to provide expert supervision for these cases. Supervision by someone who is not skilled in substance abuse is no better than no supervision at all, and at times may be worse. If no faculty member has the expertise, one might be assigned the job of finding a clinical expert to join forces to run a monthly case management seminar to review the progress of each of the residents' required cases. Ideally, this consultant should be a physician, but if the faculty member is one, it might be possible to use a non-physician consultant. Eventually, the family medicine faculty member would develop the needed expertise to run the seminar alone. It might be possible to rotate other faculty members through this responsibility as a way of enhancing the substance abuse skills of several faculty members.

Another alternative for learning clinical substance abuse skills is a block rotation in an alcoholism or drug abuse treatment program [J*Williams, 1975]. Here the family medicine resident may obtain supervision and watch experts. However, drawbacks include the lack of family physician role models, the differences in practice settings (usually inpatient and specialty care), and the short duration of the experience which limits the duration of involvement with, hence an inadequate window of observation of, a chronic disease. Perhaps the best of both worlds would be to require the block rotation early during residency at a local treatment program. If possible, it would be helpful to arrange to have the resident "pick up" several cases who needed a local primary care physician while rotating at the treatment facility, whom the resident could follow as outpatients throughout the remainder of training.

A third option for a clinical experience which has been used with medical students at the University of Minnesota, Duluth [R*Wittmers] and with family medicine residents (San Antonio, TX) as well as with impaired physicians [J*Fursch, 1978] is to actually admit the trainees to a treatment program. As consumers of care, they have the opportunity to observe first hand the affective experience of their patients who will eventually be sent there. A major drawback of this approach is finding available beds in treatment facilities at the time when the residents will be available to be "admitted". A major advantage of this approach is that it forces the self-assessment of the trainee's own experiences with and attitudes toward abuse of psychoactive sub-

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stances. Since one of the major hindrances to learning effective substance abuse skills is affliction with chemical dependence in the resident or a relative, this will be dealt with early in the process of learning about substance abuse.

A fourth option is a block rotation based at the family medicine clinic with visits to alcoholism and drug treatment programs and to AA, Alanon and Alateen meetings. Group seminars, viewing of films, and reading assignments are occupy time constructively between the visits. A confidential self-disclosure group may be utilized to discuss past and present involvement with psychoactive substances in self or family (run by the instructor and AA/Alanon members). Each resident can be assessed in terms of clinical deficits at the beginning of the rotation in order to plan individual training experiences. For example, if the resident needed practice at obtaining substance abuse histories, a series of interviews could be arranged at a local inpatient substance abuse facility, with observation by an expert. Or, the resident could arrange to interview several recovering volunteers from the community who could pretend to be patients seeking medical advice from the resident. Whenever possible, residents should pair up to do their assignments together (buddy system) to increase learning through sharing of perceptions and expertise. Readings and a special paper on some topic related to substance abuse may be added. Or a small field project may be undertaken, for instance:

1. screening 50 of the resident's own patients for alcoholism;
2. examining records of 100 clinic obstetrical patients to determine the extent of their recorded intake of alcohol, tobacco, caffeine, and other drugs;
3. developing a patient education pamphlet or tape on smoking, caffeine, or alcohol to be used in the clinic;
4. visiting a local industry to discuss alcohol/drug related work injuries and other employee problems with the employee assistance program staff and the plant doctor or nurse;
5. surveying police officers about problems with drugs and alcohol in the local community;
6. surveying local pharmacists about their attitudes and practices in relation to doctors whom they believe to prescribe too much psychoactive drugs;
7. spending a Saturday night at the local emergency room operating the breathalyzer;
8. making an annotated community substance abuse resource guide for fellow residents and faculty;
9. presenting for initial substance abuse care at a variety of local treatment programs (of course, this will have to be pre-arranged between the administrator and the teacher so as to avoid generating bills);
10. doing a physical exam on one patient recently admitted to an inpatient substance abuse program and then meeting with this patient 2-3 times per week while hospitalized to observe changes in attitudes and expectations, culminating with a home visit with the patient

- and family prior to discharge;
11. discovering the prevalence of occult substance abuse among inpatients at a community hospital at which the resident has had or will have some clinical training.

To run this block rotation takes a fair degree of time and expertise, but it is well-liked by residents (and medical students) because it is fun, creative, and tailor-made to address their interests and educational needs. It is most efficient with 6-10 residents (or students) per month, but can be done with only one. Residents of different levels and students can work together in such a rotation.

DIDACTIC FORMATS

Strictly didactic training for family medicine residents may be done by incorporating seminars on alcoholism and other substance abuse into already existing biomedical and behavioral science seminar series or into clinical management seminars [J*Williams, 1975]. Be forewarned that incorporating substance abuse only into the behavioral science series will communicate to residents that it is not a topic of "real medicine". There are numerous opportunities to incorporate a brief segment on substance abuse into many of the biomedical seminars (e.g., in a topic session on management of sexual problems, five minutes could be devoted to the impact of alcohol, inhalants and opioids on erectile and orgasmic functions). The major problem with this strategy is that it requires the full cooperation and commitment of all the teaching faculty, including outside guest speakers, in remembering to address the substance abuse issue along with the main thrust of the talk. The seminar coordinator could make this a requirement of all speakers, when appropriate to the topic, but it may take some monitoring to assure that more than token cooperation is occurring, and to be certain that opportunities are not being missed because of faculty ignorance (e.g., a session on pediatric pneumonia should mention the study showing increased incidence in children of smokers). Questions about substance abuse can be directed from the audience, as long as the questioner(s) do not become stigmatized for being the only faculty with a "fanatic" interest in substance abuse.

In presentations of clinical cases, it could be established as a departmental standard that all presentations would include a pharmacological history, whether positive or negative, whether contributory or not. This would establish the need for all who present (faculty and trainees) to have obtained this history on these cases. It would also establish the pharmacological history as a proper element of any complete database. It would provide the opportunity for experts in the audience to comment on relationships between the substance use and the salient features of the case, should they be present, even if the people presenting were not aware of the relationship. Without such data, the comments usually go over "like a lead balloon", because the presenter is embarrassed to be unaware of both the relationship and the patient's pharmacological history.

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Separate workshops on alcoholism or other substance abuse may be conducted. An example of how pertinent didactic information can be taught to third-year residents in a three-hour workshop is outlined in Table 10-2. Samples of other longer workshops are outlined in [B*Jackson, 1980, J*Liepman & Gilbert, 1979]. Various handouts and articles contained in a syllabus can be used during the workshop to augment teaching. The major drawback of this approach is getting the resident time allocated to the workshop and then mandating attendance. Such a short workshop is also limited in its scope and depth. But it can serve as a start to a curriculum, or an opportunity to tie up loose ends that could not be done in other settings. Exclusively using only the separate workshop method suffers from the lack of integration of the new knowledge, skills and attitudes into the rest of family medicine. The residents may note this as an interesting topic, but forget to use the knowledge in their clinical practices. Attitude changes may cause changes in clinical behavior, which if successful, may reinforce maintenance of the new attitudes and behaviors. New skills may likewise be tried during clinical practice with similar results. The main advantages of separate workshops is that one can plan and present an uninterrupted concentrated discourse on substance abuse while minimizing the extent of negotiation for time and cooperation with other faculty and administrators.

Didactic presentations require a careful selection of audiovisual (AV) materials and outside speakers. There are a number of AV materials available [B*Anonymous, Where the Drug Films are; B*Anonymous, 1980; J*ARF, Projection; B*Gallant, 1982; B*Hostettler, 1982; B*Rowden et al., 1983], and these can be selected to emphasize specific points. However, when selecting such a film or videotape, the learning objectives should be given prior consideration so useful and structured discussion after the film can enhance the learning experience on cognitive and emotional levels.

Providing a substance abuse elective rotation as the sole curriculum component is not advised, because many residents will complete the program without selecting this option. However, an intensive elective experience (in addition to a required basic curriculum) can be very useful. If this cannot be provided, there are some self-instructional materials available [R*Mooney; B*Schoener, 1981].

A common result of integrating teaching on alcoholism and substance abuse into a residency curriculum is increased awareness and increased identification of patients or colleagues with problems. An increased personal awareness may give rise to a better understanding of the inclination for the residents themselves to have such problems [J*Newsom & Chappel, 1980]. It is important to anticipate how such issues will be handled--a discussion group held on a regular basis for just this purpose can be a very useful experience for many residents or alternatively, use of an occasional "Balint" session might be appropriate (see Chapter 9).

THE TEACHERS

There is one crucial issue in deciding who should do the substance abuse teaching in a residency program. Many programs have behavioral scientists on the faculty who can add to the curriculum and may indeed elect to teach in this area. However, if possible, it is desirable to have a family physician involved in the teaching as a professional role model. It is also desirable to have at least one faculty member from within the program involved in teaching, rather than relying solely on outsiders, for credibility. The message to the resident when a faculty member cannot be found who can teach about a topic is that it must not be very important, valuable or relevant information or skills to have as a family physician because the faculty survive as family doctors without this expertise. Ideally, there should be at least one family physician faculty member who is an "in-house expert" for residents to consult as needed.

In choosing outside speakers, such individuals should also be encouraged to integrate affective and cognitive information into their presentations. Outside experts can offer new information or teach some refined skills, departmental faculty should be present (1) to ensure that the level and scope of the session matches the needs and resources of the residents, (2) to learn what the guest has to offer, so that eventually the departmental faculty could take over this session, and (3) to learn what from the class should be reinforced at appropriate times during clinical precepting. Recovering alcoholics and substance abusers have much to offer residents as guest speakers: they can demonstrate the impact of recovery on wellness and maturation, they can warn the resident about pitfalls to anticipate with their own patients, they can put residents in touch with local resources, and they can instill optimism in the residents who decide to confront patients who may have substance abuse problems. However, a warning must be issued that if recovering people are used in the teaching of residents, they should be counseled prior to the class to avoid using the session to vent resentments upon the residents for past bad experiences they and their friends have had at the hands of physicians.

One of the paramount problems in substance abuse curriculum design is one of faculty ignorance. This is a direct result of the history of the field of substance abuse which, until recently, has largely been developing outside of the mainstream of modern medicine [J*Noel & Colombo, 1978]. This has led to a serious knowledge, skill and attitudinal gap for most physicians, including medical school faculty [J*Filstead, Goby & Bradley, 1976; J*Jones, 1979; J*Knox, 1971; J*Liepman, Whitfield & Landeen; J*Sowa & Cutter, 1974]. While many recent medical school graduates have had the opportunity to learn something about substance abuse, many of their teachers have not had that opportunity. Consequently, there is somewhat of an inversion of roles, where the student could know more than the teacher. While on the one hand this can be a liberating experience for both student and teacher, on the other hand it can lead to dangerous teaching practices where the instructor undermines prior teachers by dis-

couraging clinical use of basic science information, by imparting pessimistic attitudes, or by suggesting harmful alternatives to constructive action. This may be done overtly, but more often is done subtly, by example. If a faculty member has a personal sensitivity to the issue of substance abuse (usually because there is an unresolved case in self or family), this may surface in a defensive manner through ridicule of the resident or student. In designing substance abuse curriculum, it is important to consider the standard of educational excellence that will be achieved, and care must be taken to avoid experiences during training that will undo what has been learned earlier. Since much of resident and student is done by faculty outside of the control of the family medicine department (in block rotations with other specialties), it may be necessary to warn the residents about faculty ignorance and prepare them to diplomatically survive any unpleasant encounters such as this without losing their commitment to high quality substance abuse care.

THE PLANNERS

As mentioned earlier, an invaluable aid is a committee of local experts to assist in planning the curriculum. However, it is essential to have both adequate faculty and resident representation on this committee. Residents themselves may be able to suggest what strategies might be most stimulating and successful.

COMMENTS: Undergraduate training

Because the emphasis of this monograph is on residency training in family medicine, the strategies for residency curriculum development have been discussed in depth. However, many of these same strategies apply to the development of an undergraduate curriculum in substance abuse as well; they will not be repeated here. On the other hand, we would like to suggest integration strategies more specific to undergraduate training.

The planning phase for curriculum development in substance abuse for family medicine at the undergraduate level should include careful consideration of the following:

1. The use of existing required courses versus new free-standing elective courses must be considered. As with the residency curriculum, incorporating components into already existing courses reaches many more students and thus is preferable to offering solely elective courses. It also allows for substance abuse to be taught as a common, treatable problem integrated with other medical illnesses, and provides an opportunity to use these problems as prototypes for discussing the general principles of family medicine.

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2. Decisions should be made as to what minimally should be taught to all medical students and, more specifically, what should be emphasized in family medicine clerkships (see Table 10-4).
3. Once decisions as to what to teach are made, thoughtful attention to integration across the four years of medical school is necessary. Clinical clerkships in family medicine often provide an opportunity for in-depth curriculum offerings, such as a seminar or case discussion, community site visit, watching videotapes [A*Substance abuse and the physician], improving diagnostic or confrontational skills [J*Clark et al, 1979; A*Liepman; J*Weinberg, 1974], or following a patient from clinic to detox center to rehabilitation program [J*Clifford, 1959; J*Coggan, Davis & Rogers, 1981; J*Confusione, Jaffe & Rosen, 1982; J*Harris & Westermeyer, 1978].
4. In approaching course committee chairpersons with curriculum materials, it is helpful to take a direct, preplanned approach. Which materials would be appropriate for the level of student training, and how might they satisfy the underlying objectives of the course [B*Davis, 1980; J*Einstein & Wolfson, 1970; B*Gallant, 1982; R*Griffin; E*Hostetler, 1982; R*Siegel; J*Stimmel, 1974]?

TABLE 10-4: Diagnosing and Managing Alcoholism

WHAT ALL MEDICAL STUDENTS SHOULD KNOW

Basic diagnostic skills:
history-taking & physical findings

Basic management strategies:
confrontation; referral to
community resources

Awareness of physicians being at
risk for alcoholism or other
drug addiction

WHAT FAMILY MEDICINE STUDENTS SHOULD KNOW IN ADDITION

Diagnosis through common complaints
in family practice setting

Diagnosis through family members;
use of family pressure to
motivate recovery

Management strategies appropriate
for the family physician in an
ambulatory setting;
provision of family support

EVALUATION: Program efficacy

This discussion of strategies for family medicine curriculum development on substance abuse would not be complete without mention of evaluation. Evaluating a program is vital for the longevity and success of the curriculum. Useful questions for an evaluation plan are outlined here.

1. What is the intended purpose and use of evaluation? Is evaluation mandated by a funding agency? If so, what questions and issues are to be addressed by the mandated evaluation? Is evaluation intended for in-house purposes in designing additional teaching units or changing existing ones? Is evaluation intended to identify changes in clinical behavior? Or will it be sufficient to merely measure resident (or student) knowledge, skills, and attitudes? If so, what level of final behavior, knowledge, skill or attitude in graduates will be acceptable to the residency program? How do faculty stack up on the evaluation? (It is unlikely that the residents, as a group, will surpass the faculty, as a group, to a significant degree.) Is evaluation intended to gauge resident, student, or faculty subjective satisfaction with the training?

2. In considering learning objectives, what evaluation methods would be most appropriate to measure the level of attainment of these objectives? The various measurement tools include observation of clinical behavior via patient care audits [B*Liepman & Gilbert, 1979] or clinical simulations [R*Corley & Fisher], patient management problems, attitude scales [B*Chappel & Krug, 1980; J*Muehler & Ferneau, 1971], knowledge questionnaires [B*Pokorny et al., 1980], surveys asking the student or resident for subjective impressions of the value of each component, and subjective evaluations by course faculty of student or resident performance. The latter evaluations are far less costly, but you get what you pay for. The most useful evaluation of a course measures change in behavior. Observation of clinical behavior in practice would be the ideal evaluation. For instance, to evaluate the value of a teaching module on clinical screening and diagnosis would be best evaluated by observing the pre- and post-module frequency of case identification in the resident's practice. In some programs this would be easily obtained from the computer, while in others it would be a major project. Patient simulations offer a controlled stimulus, the response to which can be observed by faculty. The data obtained can be multidimensional (e.g., the resident asked good questions, but did so in a crass judgmental fashion which ultimately prevented honest responses by the patient). And the data can be used not only to evaluate the training module in a summative fashion (i.e., after it is over), but repeated evaluations over the entire residency can

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provide progress reports and opportunities to alter the learning plan for the resident as performance changes. Patient management problems test the cognitive and problem-solving skills of the resident. They outshine multiple-choice knowledge tests because they more closely resemble the real clinical situation (i.e., they not only test recall of a fact, but the ability to know when and how to use the fact).

3. Where in the course offering and preexisting evaluation system might evaluation of the substance abuse module fit? How much time and resources are available to complete evaluation?
4. How should information obtained through evaluation and documentation of your efforts be disseminated? Which key people should receive it?

COMMENTS: Summary

The alteration of a curriculum is an arduous task. Each change may require time, teachers, training sites, curriculum planner consensus, well conceived learning objectives, etc. Each of these hurdles must be overcome to introduce a new training module into a family medicine curriculum. Institutional policies and personnel may interfere with rapid development of a module. Proper care to establish good relations with the medical institutions and the community agencies in which the training is being offered is essential to success. Preparing the learners to encounter controversy about what they are learning is also important to permit them to approach each training experience with an open mind and with respect. The proper balance of didactic with clinical training experiences will be determined, at least in part, by the available resources for each and the support that is available from the residency program to utilize these resources. Whenever possible, the most realistic settings must be selected so as to optimize the credibility and relevance in the eyes of the resident. Involvement of role models from the residency program faculty is very important in convincing the residents of the importance of substance abuse skills in family medicine, yet this may initially be difficult unless there are faculty with prior training in substance abuse. Usually faculty development must accompany the creation of a curriculum so as to have teachers to teach it.

AUDIOVISUAL MEDIA CITATIONS

A slight drinking problem, 1977. Rent or purchase 25 min. film from [R*Norm Southerly].

Adler, R., Endocrine and metabolic effects of alcohol, 1982. In [A*Project Cork]. Set of 25 slides and 13 pages text, \$70.

Ah! Relief: Responsible drug use. Purchase 28 min. 3/4 inch videocassette @ \$275 (Canadian) from [R*ARF].

Alcohol: Crisis for the unborn. Borrow 8 min. film or 3/4 or 1/2 inch videocassette from your local chapter of the March of Dimes; or purchase film for \$55 or videocassette for \$35 or rent film for \$10 from [R*March of Dimes].

Alcohol: How much is too much? Purchase (or rent) this 15 min. film @ \$310 (\$60) or 3/4 inch videocassette @ \$232.50 (\$50) from [R*Haseiden].

Alcohol roulette. Purchase 28 min. 3/4 inch videocassette @ \$275 (Canadian) from [R*ARF].

Alcoholics Anonymous: An inside view, 1979. 28 min. film, rent @ \$35/wk, or indefinite lease @ \$150 from [R*Alcoholics Anonymous].

Alcoholism: A chronic, treatable disease, 1979. Rent any format 60 min. videocassettes from [R*Network of CME], (order NCME #314).

Alcoholism and the physician, Project Cork of Dartmouth Medical School, 1981. 3/4 inch videocassette or 16mm film, four-part series: (1) Attitudes: 22 min.; (2) Early Diagnosis: 20 min.; (3) Confirming the Diagnosis/Initiating Treatment: 18 min.; (4) The Physician's Role in Rehabilitation: 20 min. Borrow free from [R*Modern Talking Picture Service] or purchase film @ \$950 or videocassette @ \$475 from [R*Operation Cork]. Purchase (or rent) film @ \$995 (\$100) or videocassette @ \$525 (\$70) from [R*Haseiden].

Alcoholism: Early Diagnosis and Management, 1981. 120 min. 3/4 & 1/2 inch videocassettes, purchase @ \$450 (members: \$375) or rent @ \$60 (members: \$45) from [R*AMA].

Angel Death, (Paul Newman & Joanne Woodward). Purchase this 30 min. film @ \$450 or rent @ \$70 from [R*Haseiden].

Another Chance (Sharon Wegscheider). Purchase this 32 min. film @ \$495 or rent @ \$70 from [R*Haseiden].

Bernat, J. L. & Victor, M., The neurological complications of alcohol and alcoholism, 1982. In [A*Project Cork]. Set of 64 slides and 40 pages text, \$125.

Bonkowsky, H. L., Alcohol and the liver, 1981. In [A*Project Cork]. Set of 59 slides and 31 pages text, \$115.

AUDIOVISUAL MEDIA CITATIONS

Bonkowsky, M. L., & Anderson, P. B., Alcohol: The alimentary tract and pancreas, 1981. In [A*Project Cork]. Set of 67 slides and 33 pages text, \$125.

Born with a habit, 1977. Rent or purchase 30 min. film or videocassette from [R*Mason].

Cause the Effect/Affect the Cause, 1973. Rent or purchase 23 min. film from [R*AHA].

Cheers. 5 min. 3/4 inch videocassette trigger tape about teenaged drinking is available for purchase from [R*ARF].

Ciba Symposia, Histology Slides available from [R*Ciba].

Clinical aspects of alcoholism, 1983. Series of five 30 min. 3/4 inch videocassettes: (1) Overview; (2) Neurologic complications; (3) Cardiovascular and hematologic complications; (4) Gastrointestinal complications; (5) Behavioral complications. Available through VA Medical Center libraries, or [R*VA RLRC].

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Doctor, you've been lied to, 1978. Free loan of 27 min. film from [R*Ayerst].

Don't be afraid. Obtain 11 min. slide-tape program from [R*Scripps].

Drink, Drank, Drunk (Carol Burnette). Rent 60 min. 3/4 inch videocassette @ \$55/day from [R*WGED].

Drinkers in Crisis, 1974. Rent 16 min. 3/4 & 1/2 inch videocassettes from [R*Network of CME], (order NCME #212).

Elder Ed, 1977. Rent or purchase 30 min. film from [R*GBA].

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Falling back: The dry drunk syndrome. Rent 28 min. film or 3/4 inch videocassette @ \$70 or purchase @ \$495 from [R*Hazelden].

Family secret (Liepman, M. R., & Reed, B. G.). Rent or purchase this 50 min. 3/4 inch videocassette from [R*Brown].

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Hazelden video lecture series, available from [R*Hazelden], dealing with AA steps, denial, group dynamics, etc. Write for their catalog.

How to sabotage your treatment. Purchase this 30 min. film @ \$350 or rent @ \$60 from [R*Hazelden].

If you loved me, 1977. 54 min. film or 3/4 inch videocassette, purchase @ \$250 (film @ \$450) from [R*Operation Cork], or borrow videocassette free from [R*Modern Talking Picture Service]. Purchase (rent) film @ \$495 (\$70) from [R*Hazelden].

I'll quit tomorrow, 1975. 90 min. film or 3/4 & 1/2 inch videocassette, borrow free from [R*Audiovisual Library] or preview @ \$75 or purchase film @ \$1195 or videocassette @ \$995 from [R*Johnson Institute].

It all adds up. Obtain 11 min. videocassette from [R*ARF].

It's up to you. Obtain film from [R*National Pharmaceutical].

Keep out of the reach of adults, 1974. 50 min. videocassette available from [R*CTV].

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Lisa: The legacy of Sandra Blain, 1979. Rent or purchase 22 min. film from [R*Aims], sequel to [A*The secret love of Sandra Blain, 1976] & [A*The new life of Sandra Blain].

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TITLE	TIME	BUY	RENT	GUIDE
(1) The use of alcohol.....	57 min...	\$343..	\$60..	\$3.60
(2) Problem drinking and alcoholism....	85 min...	\$515..	\$70..	\$2.85
(3) Intervention and treatment.....	87 min...	\$515..	\$70..	\$2.85
(4) Special considerations.....	87 min...	\$515..	\$70..	\$2.85

Lots of kids like us. Purchase this 28 min. film @ \$425 or rent @ \$70 from [R*Hazelton].

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Neonatal abstinence syndrome: Diagnosis, 1977. Rent or purchase 10 min. videocassette from [R*Baylor].

Neonatal abstinence syndrome: Management of the acute phase and complications, 1977. Rent or purchase 12 min. videocassette from [R*Baylor].

One day, (Dick Van Dyke). Purchase this 60 min. film @ \$595 or rent @ \$70 from [R*Hazelton].

Our Brother's Keeper, 1980. Purchase 57 min. film @ \$500 or 3/4 inch videocassette @ \$250 or 35 min. abridged version film @ \$375 or videocassette @ \$200 from [R*Operation Cork] or borrow free from [R*Modern Talking Picture Service]. Purchase film @ \$425 or rent @ \$70 from [R*Hazelton].

Project Cork of Dartmouth Medical School? Alcohol use and its consequences: A comprehensive teaching program for biomedical education. Purchase seven-part slide series from [R*Milner-

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Fenwick], prices listed separately, see also [A*Adler, 1982; A*Bernat & Victor, 1982; A*Bonkowsky, 1981; A*Bonkowsky & Anderson, 1981; A*Cornwell, 1981; A*Little & Streissguth, 1982; A*Smith, 1982].

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School days. 3 min. 3/4 inch videocassette trigger tape about suspicious adolescent peer gatherings is available for purchase from [R*ARF].

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Soft is the heart of the child, 1978. Purchase 28 min. film @ \$325 or 3/4 inch videocassette @ \$200 from [R*Operation Cork] or borrow free from [R*Modern Talking Picture Service]. Purchase film @ \$375 or rent @ \$60 from [R*Hazelden].

Some of the things that go on out there. Purchase this 30 min. film @ \$465 or rent @ \$70 from [R*Hazelden].

Resources for alcoholism treatment, (Femino, 1983). Purchase or rent this 30 min. film or 3/4 inch videocassette from [R*Brown].

Substance abuse and the physician. Series of 3/4 inch videocassettes available through [R*Office of Medical Education].

The Enabler, 1978. 23 min. film or 3/4 & 1/2 inch videocassette, borrow free from [R*Audiovisual Library] or preview or purchase film or videocassette from [R*Johnson Institute]. See also its sequel [A*The Intervention].

The Intervention, 1978. 31 min. film or 3/4 & 1/2 inch videocassette, rent free from [R*Audiovisual Library] or preview @ \$35 or purchase film @ \$495 or videocassette @ \$410 from [R*Johnson Institute]. Sequel to [A*The Enabler].

The new life of Sandra Blain, 1976. Rent or purchase 27 min. film from [R*Norm Southerly]. Sequel of [A*The secret love of Sandra Blain, 1976]; see also this film's sequel [A*Lisa: The legacy of Sandra Blain, 1979].

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RESOURCE CITATIONS

AA: see [R*Alcoholics Anonymous].

Addiction Research Foundation, Marketing Department, 33 Russell Street, Toronto, Ontario, Canada M5S 2S1; call (416) 595-6056.

AHA, see [R*American Hospital Association].

Aims Instructional Media, 626 Justin Ave., Glendale, CA 91201.

Al-Anon Family Group Headquarters, Inc., P.O. Box 182, Madison Square Station, New York, NY 10010.

Alcoholics Anonymous, Box 459, Grand Central Station, New York, NY 10163; call (212) 686-1100.

AMA, see [R*American Medical Association].

American Hospital Association, Film Library, 840 N. Lake Shore Drive, Chicago, IL 60611.

American Medical Association, Division of Marketing & Meeting Services, 535 N. Dearborn St., Chicago, IL 60610; call (312) 751-6000.

ARF, see [R*Addiction Research Foundation].

Association for Teachers of Preventive Medicine, c/o Boston University, Center for Educational Development in Health, 67 Bay State Road, Boston, MA 02215.

ATPM, see [R*Association for Teachers of Preventive Medicine].

Audiovisual Library Service, University of Minnesota, 3300 University Ave., S.E., Minneapolis, MN 55414; call (612) 373-3810.

Ayerst Laboratories, 685 Third Avenue, New York, NY 10017.

Baylor College of Medicine, Department of Psychiatry, 1200 Mour-sund, Houston, TX 77030.

BBC, see [R*British Broadcasting Corporation].

Biomedical Communications Department, Tulane University School of Medicine, 1430 Tulane Avenue, New Orleans, LA 70112.

British Broadcasting Corporation, 135 Maitland Street, Toronto, Ontario, Canada.

Brown University Center for Alcohol Studies, c/o Community Health, Arnold Lab, Box G, Brown University, Providence, RI 02912; call (401) 863-3172.

Carousel Films, Inc., 1501 Broadway, New York, NY 10036.

RESOURCE CITATIONS

Center for the Well-Being of Health Professionals, 5102 Chapel Hill Boulevard, Durham, NC 27707; call (919) 489-9167.

Ciba-Geigy Corporation, 356 Morris Ave., Summit, NJ 07901; call (212) 267-6615.

Corley, John & Fisher, Joseph V., (Inservice training exam simulated patient with alcoholism), available from Department of Family Medicine, Medical University of South Carolina, 171 Ashley Avenue, Charleston, SC 29403; call (803) 792-2411.

CTV Television Network, Ltd., Educational Film Distribution Department, Attention: Ms. Vicki Blake, 42 Charles Street, E., Toronto, Ontario, Canada M4Y 1T5.

Doctors Ought to Care, c/o Thomas P. Houston MD, National Coordinator--DOC, Floyd Medical Building, 302 Turner McCall Boulevard, Rome, GA 30161; call (404) 291-2075.

Downstate Medical Center, State University of New York, Building K, Box 9, 450 Clarkson Ave., Brooklyn, NY 11203; call (212) 735-2856.

Eli Lilly & Co., Indianapolis, IN 46206.

Emerging Technology Consultants, Inc., Box 154, Louisville, CO 80027; call (303) 447-9495.

Freer, C. P., Family health diaries help the physician to obtain an extended look at what goes on in the home in terms of stressors and their effects upon people's health. Each day the patient records brief descriptions of illness episodes and likely precipitants as well as attempts to relieve the symptoms. Sample diaries can be obtained by writing to: Charles Freer MD, Alder-moor Health Centre, Alder-moor Close, Southampton, SO1 6ST, England, United Kingdom.

Griffin, J., Substance abuse curriculum. Available from Dr. Griffin, Georgia Mental Health Institute, Room 166-A, 1256 Briarcliff Road Northeast, Atlanta, GA 30306.

GSA, Order Section, National Archives and Record Service, Washington, DC 20409; call (301) 763-1896.

Hazelden Educational Services, Box 176, Center City, MN 55012; call (800) 328-9288 or (612) 257-4010.

Hollywood Enterprises, 6060 Sunset Boulevard, Hollywood, CA 90028.

IDAA, International Doctors in AA may be able to arrange contact with a recovering doctor, c/o Lewis K. Reed, Secretary, 1950 Volney Road, Youngstown, OH 44511; call (216) 782-6216 or 788-7357.

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Intoximeters Inc., source of digital portable breathalyzer, price \$400 c/o M.R. Forrester, 1901 Locust Street, St. Louis, MO 63103; call (314) 241-1156.

Johnson Institute, 10070 Olson Memorial Highway, Minneapolis, MN 55441; call (612) 544-4165.

Lewis, D. C., & Femino, J., Alcohol teaching lab for Brown University undergraduate medical students. For more information, contact Dr. Lewis at: [R*Brown].

March of Dimes, contact local chapter to borrow film, or contact: Supply Division, March of Dimes, 1275 Mamaroneck Avenue, White Plains, NY 10605; call (914) 428-7100.

Mason, E. A., Director, Documentaries for Learning, Harvard Medical School, 58 Fenwood Road, Boston, MA 02115.

Meagher, M. D., Intervention Referral Network, Chemical Dependency Recovery Center, Hillside Hospital, 1940 El Cajon Boulevard, San Diego, CA 92104; call (619) 692-1215.

Media Library, R4440 Kresge I, Box 56, University of Michigan, Ann Arbor, MI 48109; call (313) 763-2074.

Merrell Dow Pharmaceuticals, makers of Nicorette (R) nicotine chewing gum, Cincinnati, OH 45215.

Michigan Department of Public Health, Bureau of Personal Health Services, 3500 N. Logan Street, Box 30035, Lansing, MI 48909. \$2/copy for [B*Ostrea, Chavez & Stryker, 1978].

Milner-Fenwick, Inc., 2125 Greenspring Drive, Timonium, MD 21093; call (800) 638-8652 (in Maryland call collect (301) 252-1700).

Modern Talking Picture Service, 5000 Park Street North, St. Petersburg, FL 33709; call (813) 541-7571.

Monson, L. F., Director, Bureau of Alcohol and Other Drug Abuse, Department of Health and Social Services, 1 W. Wilson Street, Room 434, Madison, WI 53702.

Mooney, Al J. III, Self-instructional curriculum on alcoholism, Willingway Hospital, 311 Jones Mill Road, Statesboro, GA 30458; call (912) 764-6236.

National Clearinghouse for Alcohol Information, PO Box 2345, Rockville, MD 20852.

National Clearinghouse on Drug Abuse Information, PO Box 416, Kensington, MD 20795.

National Council on Alcoholism, 733 Third Avenue, New York, NY 10017; call (212) 986-4433.

RESOURCE CITATIONS

National Institute on Alcohol Abuse and Alcoholism, Parklawn Building, Room 11-19, 5600 Fishers Lane, Rockville, MD 20857.

National Institute on Drug Abuse, Resource Center, Parklawn Building, Room 10A-54, 5600 Fishers Lane, Rockville, MD 20857.

National Library of Medicine, Reference Services Division, Audiovisual Resources Section, Videocassette Loan Program, 8600 Rockville Pike, Bethesda, MD 20209; call (301) 496-4244.

National Pharmaceutical Council, 1030 Fifteenth Street NW, Washington DC.

NCA, see [R*National Council on Alcoholism].

NCALI, see [R*National Clearinghouse for Alcohol Information].

NCDAI, see National Clearinghouse on Drug Abuse Information.

Network of Continuing Medical Education, 15 Columbus Circle, New York, NY 10023; call (212) 541-8088.

New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021.

NIAAA, see [R*National Institute on Alcohol Abuse and Alcoholism].

NIDA, see [R*National Institute on Drug Abuse].

NIDA Resource Center, Audiovisual Loan Service, 5600 Fishers Lane, Parklawn Building, Room 10A-54, Rockville, MD 20857; call (301) 443-6614.

Norm Southerly Productions, 1709 E. 28th, Long Beach, CA 90806.

Office of Medical Education, Anderson Medical Sciences Building, School of Medical Sciences, University of Nevada, Reno, NV 89557.

Operation Cork, 8939 Villa La Jolla Drive, San Diego, CA 92037; call (714) 452-5716.

Rosett, H., Department of Psychiatry, Boston University School of Medicine, 51 Barnstable Road, West Newton, MA 02165; call (617) 527-0577. Dr. Rosett has developed a slide show on the Fetal Alcohol Syndrome with an accompanying reference guide which can be used for a variety of presentations from lay to professional or scientific audiences.

SAAETP, see [R*Southern Area Alcohol Education and Training Program].

Schoener, E. P., Department of Pharmacology, Wayne State University School of Medicine, 540 E. Canfield, Street, Detroit, MI 48201; call (313) 577-1570.

FAMILY MEDICINE CURRICULUM GUIDE TO SUBSTANCE ABUSE

Siegal, M. A., Department of Medicine in Society, Wright State University. An integrated multispecialty curriculum on substance abuse. Available from Dr. Siegal, Department of Medicine in Society, Wright State University School of Medicine, Dayton, OH; call (513) 873-0350.

Society for Teachers of Family Medicine, c/o American Academy of Family Physicians, 1740 W. 92nd Street, Kansas City, MO 64114; call (800) 821-2512.

Southern Area Alcohol Education and Training Program, Inc., 4875 Powers Ferry Road, NW, Atlanta, GA 30329.

SPIT, Scientific Products, Instruments & Technologies, Inc., P.O. Box 55, Manchaug, MA 01526. This company makes an instrument helpful in determining free drug levels in patients who are on multiple drugs simultaneously so that their total drug levels would not accurately reflect free drug levels.

STFM, see [R*Society for Teachers of Family Medicine].

Time-Life Video, 100 Eisenhower Drive, Paramus, NJ 07652; call (800) 526-4663.

US Government Printing Office, Washington, DC 20402.

Veterans Administration Regional Learning Resources Center, St. Louis, MO; call (314) 487-0400 ext. 484.

Vista Hill Foundation, 3420 Camino del Rio North, Suite 100, San Diego, CA 92108.

Walt Disney Educational Media, 500 S. Buena Vista Street, Burbank, CA 91500.

Wittmers, L., University of Minnesota (Duluth), School of Medicine, Duluth, MN 55812; call (218) 726-8551.

WOED, Attention: Karen Rothrock, 4802 Fifth Avenue, Pittsburgh, PA 15213; call (412) 622-1300.